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The University of Nottingham

Modelling crop diseases for food security

Masoud Sulaiman Abood Al-Azri, MSc.

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ABSTRACT

Global crop production is affected by seasonal and climatic variations in temperature, rainfall patterns or intensity and the occurrence of abiotic and biotic stresses. Climate change can alter pest and pathogen populations as well as pathogen complexes that pose an enormous risk to crop yields and future food security. Crop simulation models have been validated as an important tool for the development of more resilient agricultural systems and improved decision making for growers. The Agricultural Production Systems Simulator (APSIM) is a software tool that enables sub-models to be incorporated for simulation of production in diverse agricultural systems. Modification of APSIM to incorporate epidemiological disease model for crop growth and yield under different disease intensities has few attempts in the UK or elsewhere. The overall aim of this project is to model disease impact on wheat for improved food security in two different agro-ecological zones.

The incidence of wheat diseases between 2009 and 2014 in two different agro-ecological zones, UK and Oman were compared. Most of the fields surveyed in Oman and UK were found to have at least one disease. Leaf spot was the most prevalent foliar disease found in Omani fields while *Septoria* was the most common foliar disease in the UK. Fusarium followed by eyespot and ear blight represents the most common diseases of stem and ears in UK winter wheat between 2009 and 2014. However, in Omani wheat Fusarium causing stem base and loose smut of ears were the most common. Eyespot was not found in Omani winter wheat and this may relate to the high temperature during winter

in Oman. This study discussed the first work on the occurrence of fungal diseases and their pathogens in Oman and the influence of agronomy factors. Large numbers of pathogenic fungi causing symptoms were found to be prevalent in wheat fields in Oman. Isolation from six symptomatic wheat varieties resulted in 36 different fungal species. *Alternaria alternata* was the most frequently isolated pathogen followed by *Bipolaris sorokiniana*, *Setosphaeria rostrata*, and *Fusarium equiseti*. Results also showed some agronomic practices influenced disease incidence. Mechanical sowing method and time of urea application were found to influence leaf spot disease.

An investigation into the recovery of treatment cost for eyespot control through yield and the effect of fungicide treatment on risk showed that all fungicides apart from (epoxiconazole) Opus at 1 L ha⁻¹ were found to be worth the costs, either under high disease pressure (inoculated sites) or naturally infected sites. For the risk averse manager fungicide treatment would be worth the cost as it would reduce the higher level of disease and consequently minimise associated yield losses.

In this work, disease models were built to predict the disease development and yield loss in relation to crop phenology using results from previous literature on conditions favouring sporulation, infection and disease development and severity. Analysis of 461 data sets showed that climatic conditions and agronomic factors significantly influenced disease development either positively or negatively in all models. The application of a range of fungicides at GS31/32 reduced disease significantly at GS39 in comparison to

epoxiconazole alone. Disease severity at GS39 decreased yield only slightly by 2.2% whilst only (prothioconazole) Proline 275 increased yield significantly with almost 30% yield increase.

The performance of the APSIM wheat model to simulate phenology, leaf area index, biomass and grain yield of two winter wheat varieties (Okley and Cashel) was evaluated under UK conditions and the previously developed eyespot disease were linked with APSIM. Generally, APSIM poorly predicted the phenology, LAI, biomass and yield of winter wheat grown under UK conditions. The linked eyespot disease models with APSIM simulated an adequate level of disease predication at GS12/13 (9.6%), GS31/32 (1.3%) and GS39 (12%).

Overall, the link between eyespot epidemiological disease models and crop growth model has successfully provided the basis for further development of the model and enhance crop growth simulation. Moreover identification of main diseases threatening wheat production in Oman can help to plan for future research, to assess the economic importance and to contrast environment models for yield loss.

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ABBREVIATIONS

AHDB-HGCA	Home-Grown Cereals Authority
AIC	Akaike's Information Criterion
APSIM	Agricultural Production Systems Simulator
AUSM	Cropping system model for operational research
BIC	Bayesian Information Criterion
BPMN	Business Process modelling Notation, bpmn.org
CERE-Wheat	Model of the growth, development, and yield of spring and winter wheat
CH ₄	Methane
CMP	Common Modelling Protocol
CO ₂	Carbon dioxide
CROPGRO	Crop components and growth change based on farming system and climatic conditions
CV	Coefficient of variation
DDM	Disease development model
df	Degrees of freedom
DI	Disease incidence/index
DNA	Deoxyribose nucleic acid
DSM	Disease severity model
DUL	Drained Upper Limit
DYMEX	Computer software that allows the user to build and run computer models which describe the lifecycles and management of biological organisms
EcoCrop	FAO-EcoCrop database model
exp	Exponential value
FAO	Food and Agriculture Organisation of the United Nations
AquaCrop	Water defects crop model
FG	Fungicides treatments
FHB	Fusarium head blight
GAI	Green area index
GLAM	General large area model
GLMs	Generalised linear models
GPS	General position system
GRAZPLAN	Pasture and animal production
GS	Plant growth stage (Zadok's)
ha	Hectares
HI	Harvest index

IP	Infection potential
IPM	Infection potential model
ITS	Internal transcribed spacer region of the ribosomal DNA
kg	kilograms
KL	Rate of maximum daily water uptake per day
LAI	Leaf area index
L	Liters
LL15	Water content at 15 bar
LSD	Least significant difference
MAF	Ministry of Agriculture and Fisheries
MBC	Methyl benzimidazole
MSE	Mean squared error
NaOCl	Sodium hypochlorite
NCBI	National Centre for Biotechnology Information
ND	Number of days
NO ₂	Nitrous oxide
ns	Not significant
OA	<i>Oculimacula acuformis</i>
OY	<i>Oculimacula yallundae</i>
P	Probability value
PAWC	Plant Available Water Capacity
PCR	Polymerase chain reaction
PDA	Potato dextrose agar
PERFECT	Productivity, Erosion and Runoff Functions To Evaluate Conservation Techniques
REML	Residual maximum likelihood REML
RH	Relative humidity
S.S	Sum of square
SAT	Saturation point
SDs	Standard deviations
SDW	Sterile distilled water
SED	Average standard error of difference
SEM	Standard error of the mean
SI	Severity incidence/index
SMD	Soil moisture deficit
SMN	Soil mineral nitrogen
Soil-N	Model nitrogen model that balance available soil carbon and nitrogen as well as their dynamics
Soil-Wat	Model Water balance model that distributes water throughout the soil profile

t	Tones
TAG	The Arable Group research
T_d	Mean daily temperature
T_{dmax}	Daily maximum temperature
T_{dmin}	Daily minimum temperature
TGW	Thousand grain weight
TR	Total rainfall
TT	Thermal time
UKCIP02	Scenarios predicting UK temperature/rainfall under high- and low-CO2 emission
Vensim TM	Weed seed bank model
WS	Wald statistics

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Chapter 1

1. INTRODUCTION AND LITERATURE REVIEW

1.1. FOOD SECURITY IN GLOBAL AGRICULTURE

Food security is predicted to be a serious future challenge due to increasing global population and income per capita in developing countries as well as scarcity of natural resources such as land and water (Duveiller et al., 2007). According to James (1998), at the beginning of 1990s there were more than 800 million people among various regions of the globe unable to obtain sufficient food to satisfy their nutritional needs. This number increased to 1.02 billion people in 2009 due to reduced availability of adequate food (Anonymous, 2010). Indeed, von Braun and Torero (2009) linked food security with access to sufficient safe food more recently as a result of the spike in the prices of the food commodities around the globe in 2007 and 2008. The Food and Agriculture Organisation of the United Nations (FAO) (1996) defines food security “when all people, at all times, have physical and economic access to safe, sufficient, and nutritious food to meet their dietary needs to ensure an active and healthy living”.

The major factors affecting long-term food security are climate change and in some cases increased biofuel production from food crops. Population and income growth have intensified the pressures on natural resources (Alcamo et al., 2005) and factors such as economic recession, political unrest, war, climate change, poverty and unemployment can influence food availability and access. Individual factors or combinations can thus cause “hotspots” of food insecurity around the world (Scholes & Biggs, 2004). Drought and conflicts among the communities have been known to cause food insecurity in most developing

countries (Brinkman & Hendrix, 2011). On the other hand, rising water demand together with increasing population and urbanization also affect food production.

Food is a basic human need and one of the most fundamental human rights is access to enough food to enable a healthy and active lifestyle. Food energy deficit therefore has been used as a measure of food insecurity. Von Braun (2007) demonstrated that economically poor countries have inadequate social, institutional and cultural systems to adjust to unpredicted disturbances such as climate change and water scarcity culminating in severe impact in food security. Moreover, climate instability as well as water scarcity, land degradation and pests and crop diseases can have direct influence on farm production and consequently on food security in developing countries (Parry et al., 2004). Thus, in developing countries improving crop production is one of the most efficient ways to reduce poverty and increase food security.

1.2. CHALLENGES TO FUTURE FOOD SECURITY

Achieving security of food supply under changing climate is a major challenge of the 21st century given that an increase in food demand of 70 to 100% is projected by 2050, a high percentage of which will need to be met by the main staple cereal crops (wheat, rice and maize). Observations and climate projections suggest that major threats to cereal production and food security are likely to arise through increased frequency and severity of extreme weather events. Such events include seasonal variation in temperature and changes in rainfall patterns and intensity, which would result in reduction, and possible

failure, of crop production (Mishra et al., 2008). This has been demonstrated in recent years by the simultaneous occurrences of adverse weather events in important agricultural production regions across the world (Lobell & Gourdji, 2012). These events are implicated as the main causes of increased food supply shortages and spikes in food prices (Wheeler & Von Braun, 2013).

1.2.1 INCREASING GLOBAL POPULATION FOLLOWING THE GREEN REVOLUTION

The world population was estimated as 6.94 billion people in 2011, expected to increase to approximately 9 billion by 2050 (Figures 1.1 & 2.1) (van der Mensbrugghe et al., 2009). The danger with this rapid increase in the population is that food production at subsistence level, mainly in some countries in Asia and Africa, is unlikely to keep up with the rate of growth in the context of limited natural resources such as water and land (Shiklomanov, 1991). The growth in population means that more land is required for settlement thus limiting the land available for farming. The demand for cereals in both developing and developed countries is projected to grow from nearly 2.1 billion tonnes in 2005 to 3 billion tonnes by 2050. Thus to feed nearly 9 billion people in 2050, food production will need to grow by almost 70% between 2005 and 2050. This suggests that food production in the developing countries alone will need to double.

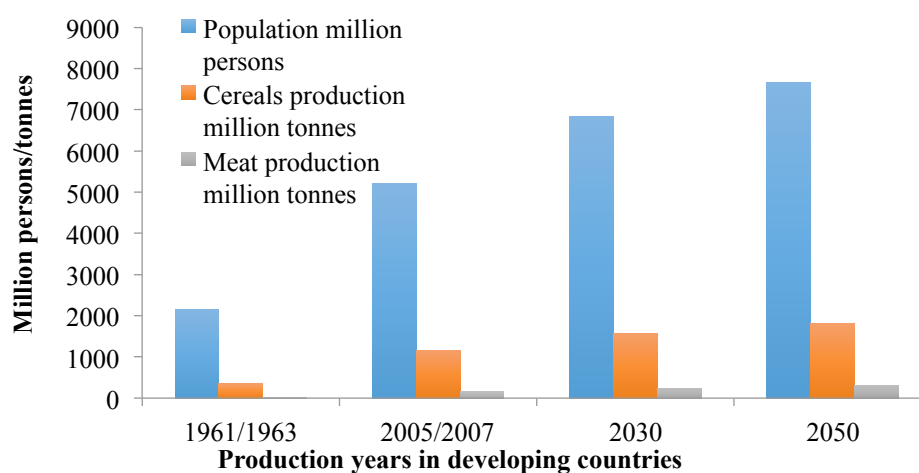


Figure 1-1: Historical and projection population, cereals and meat production in developing countries (adapted from van der Mensbrugghe et al., 2009). Rapid increase of population from just 2000 million persons in 1961 projected to be 9000 million persons by 2050 accompanied by slow growth in cereal and meat production in developing countries.

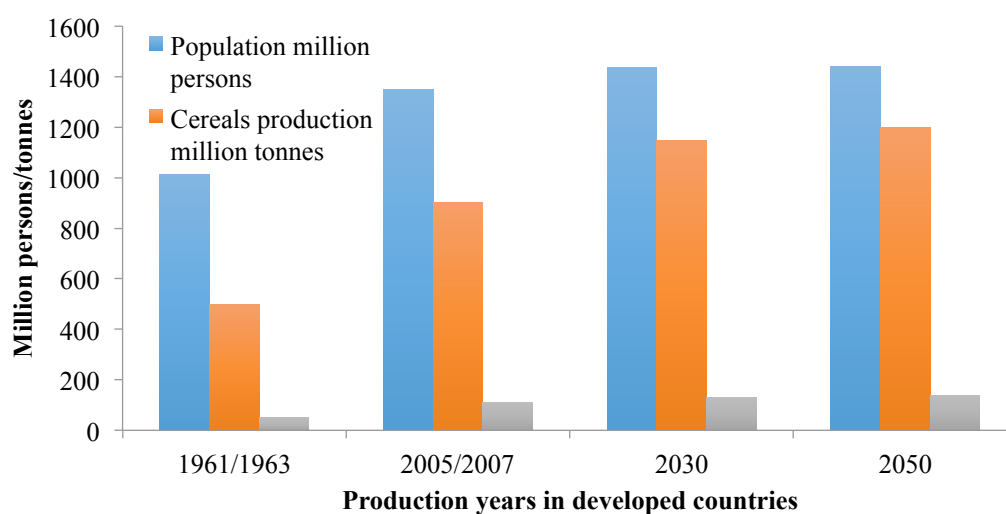


Figure 1-2: Historical and projected population, cereals and meat production in developed countries (adapted from van der Mensbrugghe et al., 2009). The demand for cereal and meat in developed countries is projected to grow from nearly (500 cereals, 52 meat) million tonnes in 1962 to (1200 cereals, 138 meat) million tonnes in 2050.

The Green Revolution has helped to feed the increasing population of the world through the development of high-yielding crop varieties, irrigation

infrastructure, innovation of management techniques, distribution of hybrid seeds, synthetic fertilizers, and pesticides to farmers (Islam et. al., 2012). However, the Green Revolution advancements seem to have been used less in helping developing countries to meet the demand for food. In Africa agricultural production decreased by 5-10% per capita from 1980 to 1995 (Sanchez et. al., 1997). In contrast, in the developed countries the wide application of biotechnology for plant breeding in agriculture has aided the production of plants that can use soil more efficiently and resist constraints like disease, drought and salinity (Chen & Kates, 1994). Increased uses of fertilizer and pesticides and crop improvement (Ingram et. al., 2008) have had a direct effect on crop yields, leading to increased food production (Ainsworth & Long, 2005). Thus in Europe and in the USA the average yield of maize, rice and wheat since the Green Revolution have increased by 61, 54 and 41 kg ha⁻¹ year⁻¹, respectively (Ainsworth & Long, 2005). Moreover, improvements in farming practice as well as crop management and better protection against pests and diseases have contributed further to a significant rise in wheat production over the last 40 years (Chakraborty & Newton, 2011).

1.2.2 CLIMATE CHANGE

Changes in climate due to global warming have become a major concern among the global organizations and governments. For instance, the weakest economic region is the most vulnerable to climate change (Christensen et al., 2007). Also areas located in the low latitude and less developed regions face greater risk to be affected by changing climate. This is mainly based on the

social, economic as well as the political constraints that determine the capacity and the ability of systems to cope with external stressors and the associated food insecurity that would accompany a changing climate (Brown, 2009; Cooper et al., 2008). Countries that are dependent on natural resources for food production via agriculture, pastoralism and fishing are particularly vulnerable to climatic changes regardless of whether they are in the developed or the developing world.

The changes in the climatic conditions, and particularly global warming, have led to long periods of drought to the detriment of agricultural activities. According to Cooper et al. (2008) and Jones and Thornton (2009), the predominance of rain-fed agriculture has resulted in a food system that is highly sensitive to the changes in the environmental conditions. The evidence of climate change can be clearly seen in the case of Asia, the highest populated area in the world, where 25% of the world's cereal production is projected to be affected if changes in rainfall occur that lead to drought or flooding (Chakraborty & Newton, 2001). Gulf countries are also likely to be rapidly affected by climate change in terms of extreme temperatures since they are already in an arid region. Oman for instance, is highly vulnerable to climate change, as it is one of the most water scarce countries, and less and more erratic precipitation due to changing climate will also effect the balance in water supply and demand, which will likely worsen the country drought and desertification incident (Mushtaque & Choudri, 2012).

Climate change on the other hand is altered by increasing agricultural production. Due to increase in the use of fertilizers and pesticides in crop cultivation in the last century the concentration of carbon dioxide (CO₂), nitrous oxide (NO₂) and methane (CH₄) gases in the atmosphere increased by 25, 16 and 100% respectively (Chen, 1990; Rosenberg and Scott, 1994; Houghton et al., 2001; Hoffert et al., 2002). Several studies have provided evidence of the negative impact of global warming on crop production. A study about implications of global changes for natural and managed terrestrial ecosystems found that wheat cropping duration and yield has decreased with increasing global temperature. The same study showed that the rise of one temperature degree above 32.8°C would result in a yield reduction of 5% in rice (Walker and Steffen, 1999). Kurukulasuriya et al. (2006) investigated the impact of climate change on African agriculture and concluded that African farms are sensitive to climate change. It was estimated that temperature increases of 1.9°C in dry land crops and 0.5°C in irrigated crops will have elasticity of response to the farmer's income. Such increases in temperature would reduce crop yields and encourage pests and diseases; thus the costs associated with farming would increase and farmer income would decline.

1.2.3 SCARCITY AND COST OF RESOURCES

The widespread degradation and the heightened scarcity of land and water resources have placed most of the food systems in the world at risk (Figure 1.3), thus posing a major challenge on the ability of these systems to effectively and efficiently feed the growing population of people in the world

(Berndes, 2002). Uses of such resources for non-food applications, like urban and industrial development as well as production of biofuel, will detract from potential world food supply.

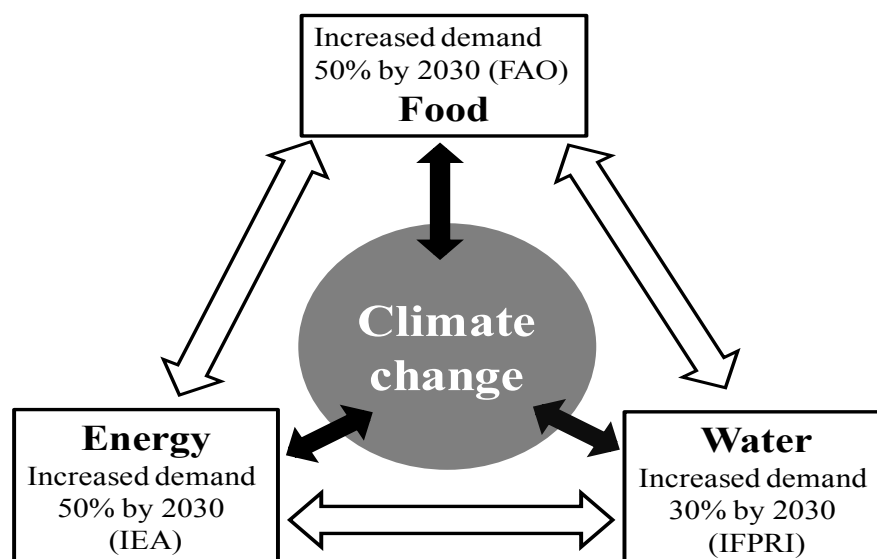


Figure 1-3: Challenges of climate change and interactions of water, food and energy by 2030 (adapted from Islam et al. 2012). Under climate change challenges in 2030 the demand of food is projected to increase by 50% accompanied with 50% increase in energy demand and 30% increase in water demand.

Globally, agricultural cultivation only occurs on 12% of the total land (Schultz & de Wrachien, 2002). Land degradation due to salinization, desertification, soil erosion and deforestation will pose further challenges in increasing food production from available arable land. For instance, FAO (2003) reported that there has been a reduction of almost 13% of usable agricultural land and 4% of pasture in the last 50 years. The high cost of molecular breeding, genetic engineering and molecular diagnostic tools has impeded the contribution in improving plant yields and consequently reducing hunger in highly populated

areas of Asia and Africa (Messer & Heywood, 1990). In addition water scarcity has increased across the world, in particular in developing countries. Agriculture uses almost 70-80% of the freshwater from the global river system (Molden et al., 2007), and growing agricultural needs would further deplete this resource.

1.2.4 PESTS AND DISEASES

Food security is influenced by climate change, which in turn can affect the incidence of pests and diseases in major crops (Gregory et al., 2009). Potential yield has been the target in many assessments of climate change effects on crops, but actual yield can be impacted highly by pests and pathogens (Gregory et al., 1999). Pests and diseases can damage the growth and yield of crops that provide food for humans and as such threaten food security. According to Oerke and Dehne (2004) insect pests are the most important crop yield reducers and are mainly favoured by monoculture and the intensive application of fertilizers. Under changing climate pests that usually occur at low densities, may be able to spread widely and reach higher more damaging population densities. For instance, aphids, the global key pests of agriculture, horticulture, and forestry, are likely to respond to climate change because they have low temperature threshold for development, short life cycle and high ability to spread (Sutherst et al., 2007).

Climate change can also cause changes in pathogen complexes in turn altering the impact on crop yield, safety and quality. Fusarium head blight (FHB) disease in wheat is a good example where disease incidence and severity has

been altered due to changes in climate (Chakraborty & Newton, 2011). Due to prolonged wet weather periods, FHB re-emerged in the northern Great Plains and central USA between 1998 and 2000, causing yield loss and grain price reduction estimated to total \$2.7 billion as a result of reducing grain quality and safety (Goswami & Kistler, 2004). Beside yield losses associated with FHB infection, economic losses are associated with the production of, trichothecene mycotoxins and oestrogenic zearalenone in infected host tissue, which are harmful to humans and animals.

Disease development, host physiology and host resistance can be altered by climate change. Plant canopy size and density can increase significantly by higher levels of CO₂, which in turn results in higher nutritional quality and a greater biomass (Manning & Tiedmann, 1995). Such changes to the plant however may promote foliar diseases such as rusts, powdery mildews, leaf spots and blights, particularly when excessive humidity exists in the canopy (Coakley et al., 1999). Furthermore, many pests and diseases are capable of relatively rapid genetic changes. Altered environmental conditions that emerge due to climate change may enhance their ability to invade new areas or alter their seasonal patterns and abundance and as such threaten crops in locations which would otherwise not have preventative measures in place (Clements & Ditommaso, 2011). Survival, development, reproduction and dispersal of plant pathogens are dependent on climate to a certain degree, and accordingly shifting weather patterns may influence pathogen epidemiology and undermine any crop protection that is currently employed.

Despite recent advancements in breeding and integrated pest management, crop losses due to pests and diseases are still high, reaching over 50% in the major crops under favourable conditions such as high temperature and high rainfall (Oerke, 2006). Oerke (2006) analysed losses of 6 major crops between 2001 and 2003 and found that the average loss in wheat and cotton due to plant diseases was 29%, while in potato the loss was 40%. Apart from pre-harvest crop losses due to pests and disease there it is estimated that up to 10% further loss can occur due to post-harvest diseases (Strange & Scott, 2005). Smil (2000) estimated that the overall waste in available food “from field to fork” was almost 25-55%, considering the combined pre- and post-harvest losses. The variation in loss between locations and seasons is influenced by the variation in climate, which in turn influences the incidence and severity of crop pests and diseases (Flood, 2010).

Accurate data on the yield losses caused by disease in developing countries are often absent or difficult to obtain. However, this is not the case in developed countries, for example in the UK, where more information about disease and estimated losses is available. Effects of climate change on plant disease epidemics have been investigated less often. More work is required to understand the impact of climate change on the interactions between crops and diseases, and the outcomes of these interactions on crop production. A recent report from the UK Government Office for Science stated that development of optimal disease management strategies under predicted climate change scenarios are needed for agriculture to consider the impact from future threats of plant disease epidemics (Brownlie et al., 2006). The impacts of pests and

diseases on yield in current conditions are well known, but the consequences of climate change on pests and diseases are complex and, as the preceding descriptions attest, are still only imperfectly understood.

1.3. ECONOMIC PERSPECTIVES

Biotic stresses can decrease yield, increase production cost and limit food storage and market. In addition, pests and diseases can affect crops and can cause large economic losses and threaten food security. The national economy of countries dependent on the production of a single crop, which may be reduced by pest and disease, are under higher food insecurity (Walker, 1983). Therefore, management of disease should not only consider the epidemiology, but also the economics of crop protection. Agriculture is characterised by a large exposure to risk, high levels of uncertainty in output value and with the fact that decision consequences are not always known when management decisions are made. Variability in prices and yield is the biggest source of risk, whilst technology and policy change also have an impact (Moschini & Hennessy, 2001). Furthermore yields are highly variable and can be affected by a range of factors, including pests and diseases that can play a major role in effecting this outcome.

For instance, eyespot, a stem base disease of wheat, can cause significant economic impact in winter wheat in England and Wales (Hardwick et al., 2001). This disease gained more attention in Europe after methyl benzimidazole (MBC) fungicides were rendered ineffective and the pathogen gained resistance to them (Brown et al., 1984; King & Griffin, 1985). It has

been difficult to quantify the economic impact of eyespot disease due to difficulties in visually diagnosing the disease in the presence of other stem base diseases such as brown foot rot and sharp eyespot (Polley & Turner, 1995). Potential losses from this disease have been found to vary depending on the severity of infection. For example, slight infections have been shown to cause little loss in ear weight (Ray et al., 2006), whilst under moderate or severe infection, yield has been found to be reduced between 10% and 36% respectively (Clarkson, 1991). The values of yield loss vary between 0.5% and 2.2% of the total national yield (Hardwick et al., 2001). This finding was supported by Cook et al. (1991), who investigated yield loss in winter wheat between 1985 and 1989 in England and Wales, showing that the national yield reduction due to eyespot was over 250,000 tons of wheat per year. Management of the disease in the case of eyespot was considered mostly in respect to yield loss, but the economics of the treatment cost, or the benefit in terms of gross margin have not been fully considered.

1.4. SIMULATION OF CROP PRODUCTION AND MODELLING OF LOSSES DUE TO PESTS AND PATHOGENS

Since the 1990s, crop simulation has been an important tool for supporting decision making in crop production. Most crop models are blends of mechanistic approaches and empirical assumptions, therefore continuous research is needed to improve the capture and accuracy of data during extreme adverse weather as would be expected due to climate change (Challinor et al., 2003). Modelling can help to understand better more complex interactions

between different components in agricultural systems which also aids in predicting the appropriateness of management strategies (Challinor et al., 2003).

Existing crop models rely on two different approaches. The first approach is to quantify crop growth and development as well as climatic conditions, for example EcoCrop developed originally by Hijmans et al. (2001), Agricultural Production Systems Simulator (APSIM) developed by McCown et al. (1996), the general large area model (GLAM) developed by Challinor et al. (2004) and the (CROPGRO) model developed by Boote & Jones (1998). The second approach is based on an experimental model that is concerned with only one variable. Particularly, the basic purpose of different crop simulation models is to model how plant growth and yield are affected by changes in the environment. Therefore, the predictions generated assist our understanding about the future impact of climate change on crop production (Jamieson et al., 1998). The input data in any model must be of adequate quality, to reduce the uncertainty in the output result. Moreover, the clarity of the result and explanation will be reduced by excessive complexity of input data (Passioura, 1996).

APSIM is one of the most used simulation models for cropping systems in the dry lands. APSIM allows integration with other models such as those use for pasture and animal production for example GRAZPLAN (Moore et al., 1991), utilised in the Mediterranean and temperate regions of Australia (McKeon et al., 1990) and used in subtropics and tropics. This integrative function is very

important as it facilitates the incorporation of sub-models within APSIM in order to make more complex simulations. Thus the model can simulate a farming system where there is an uncertain or insufficient rainfall as well as decreased soil fertility and soil structure economically impacting future crop production. Another crop simulation approach is CROPGRO, which is a computerized model that estimates crop components and growth change based on farming system and climatic conditions (Boote et al., 1998). This model can also simulate soil saturation, organic matter and nitrogen balance. Moreover, it can constitute genetic differences among species and cultivars of some legume crops, however it is not able to simulate the growth of cereal crops. This model is more reliable to identify homogeneous crop region rather than heterogeneous as it is less justifiable in spatial aggregation of inputs over heterogeneous land because it includes more process of non-linearity (Basso et al., 2001; Jones & Barnes, 2000; Guerif & Duke, 2000).

EcoCrop originally developed as DIVA-GIS created by Hijmans et al. (2001) was given this name because it is using the FAO-EcoCrop database (2000). This model is more applicable to wider geographical areas rather than a single location as it allows spatial analysis of including landscape features; however, it requires adequate representation of current climate for optimum spatial resolution. FAO-AquaCrop (Steduto et al., 2009; Raes et al., 2009), is used for the prediction of the impact of water deficits on crop production for major field and vegetable crops using low input data and parameters such as biomass, soil evaporation, crop transpiration and final yield (Raes et al., 2009). This model provides improved balance between accuracy, simplicity and robustness as

well as its parameters are easy to understand and use with clear outputs and details. One drawback is that AquaCrop is using harvest index (HI) parameters to estimate yield. However, to avoid uncertainty and difficulties, which may exist with the separation process, the separation of biomass into organs like leaves or roots is not calculated. On the other hand, General large area model (GLAM) (Challinor et al., 2004) aims to unite the advantage of modelling using low input data over large area (Fischer et al., 2002), a process-based approach modelling. Equations and parameter values of different crops have been developed within the model to permit operation in larger number of annual crops. This highly parameterised model has forty parameters, twenty of them being crop specific and the rest of them can vary spatially (Challinor et al., 2004).

Combinations of models that focus on future climate simulation, crop growth and empirical disease measurements have been developed for many diseases such as phoma stem canker of oilseed rape (Evans et al., 2008) and light leaf spot (Welham et al., 2004). UKCIP02 scenarios predicting UK temperature/rainfall under high- and low-CO₂ emission scenarios for the 2020s and 2050s were combined with a crop simulation model for yield of fungicide-treated winter oilseed rape and weather-based regression models for severity of phoma stem canker (Evans et al., 2008) epidemics to investigate crop-disease-climate interactions (Butterworth et al., 2010). Yields of fungicide-treated oilseed rape crops in 2020s and 2050s were predicted with the greatest yield increase of 15% in eastern Scotland. The same model predicted that epidemic severity of phoma stem canker and climate change will contribute to yield loss

of moderately susceptible untreated crops by up to 50% (264,000 t/ha) in southern England (Evans et al., 2008; 2010; Butterworth et al., 2010).

Although a considerable amount of literature has been published on modelling approaches that aim to develop crop simulations to understand optimal conditions to enhance crop output, few attempts have been taken to modify the simulation software to incorporate epidemiological disease modelling on crop growth and yield under different disease intensities in the UK or elsewhere. APSIM was chosen for the purpose of this study over the Sirius model (that has been evaluated to simulate the growth of wheat crop in the UK) (Jamieson et al., 1998), because of two important reasons; i) APSIM can simulate wheat growth and yields under any crop growing conditions and ii) the multi-point features within APSIM that allows it to simultaneously simulate multiple points in space and the interactions between them as well as the input and output features that simplified communication between multiple models which does not exist in the Sirius model.

1.5. AIMS AND OBJECTIVES

The overall aim of this project is to model disease impact on wheat for improved food security. The main objectives are:

- 1) To compare the incidence of wheat diseases between 2009 and 2014 in two different agro-ecological zones, the UK and Oman. Also, to identify the

main disease threats and quantify their impact on wheat production in Oman using data of diseases in Omani wheat collected in a 2014 survey.

- 2) To improve economic decision-making relating to different eyespot management strategies by i) assessing whether treatment cost of eyespot control is recovered through yield response of the crop and ii) to assess the effect of fungicide treatment on risk using the same data on eyespot disease and fungicide efficacy carried out between 2004 - 2014 in the UK.
- 3) To develop conceptual epidemiological disease model for the prediction of yield loss in wheat. Collected data from wheat experiments on eyespot disease and fungicide efficacy carried out between 2004 and 2014 in the UK were used for this work.
- 4) To evaluate APSIM for its ability to simulate winter wheat development and yield under UK conditions when incorporated with an epidemiological model for eyespot disease.

Chapter 2

2. THE OCCURRENCE OF FUNGAL DISEASES IN OMAN AND UK BETWEEN 2009 AND 2014 AND CHARACTERISATION OF THE PATHOGENS IN OMANI WHEAT

2.1. INTRODUCTION

Wheat is one of the most important cereal crops worldwide with a total production of 734.2 million tonnes in 2015/16 (FAOSTAT, 2016). Wheat production under arid conditions as in Oman is largely limited by water availability and domestic consumption exceeds production. The cultivated area in 2014 was 660 hectares and the total production was 2100 tonnes (FAOSTAT, 2015). The average crop yield has increased from 1.2 t/ha to 3.8 t/ha due to the introduction and adaption of higher yielding varieties and more efficient irrigation systems (Curtis et al., 2002). Whilst major research efforts have focussed on improving yields through improved crop breeding and irrigation systems, accurate data on yield losses due to pest and diseases in arable crops in developing countries is often absent (Kamal et al., 2010). Worldwide it has been estimated that up to 29% of wheat yield is lost due to diseases (Oerke, 2006). However, in Oman there is limited information on the main diseases of wheat crops and the pathogens associated with them, while in the UK, incidence and severity of diseases of winter wheat have been recorded since 1970.

The position of Oman between north eastern Africa and north western Asia, a region where in 2007 a new virulent strain of wheat stem rust, race Ug99 (Singh et al., 2007) was confirmed prompted the first surveys of diseases in wheat. Rust caused by *Puccinia triticina* on wheat was first reported by Deadman (2007). Al-Sadi (2010) isolated *Bipolaris sorokiniana* (*Cochliobolus sativus*) and *Alternaria alternata* from the seed of two different field grown

varieties of wheat and investigated the influence of seed-borne *B. sorokiniana* on severity of root and crown rot of wheat and barley.

There is further lack of information on the agronomy aspect of wheat production and its consequence for disease occurrence and pathogen predominance within standard farming practices in Oman. Omani wheat is cultivated only under irrigated conditions predominantly on sandy loam soils of low fertility (MAF, 1993). The crop is grown in rotation with maize, sorghum and alfalfa as well as other minor crops. Previous crop residue is usually removed for livestock. However, some returns through mixtures with compost or manure are practiced in isolation. Residue burning is still practised, whereas harvesting is mechanised. Seed treatment is not practiced prior to sowing. Most nutrients are supplied as livestock manure prior to sowing although artificial nitrogen at 150kg/ha, phosphorus at 90kg/ha and potassium at 60kg/ha are typically applied during the growing season (Al-Lawati & Nadaf, 2001). Fungicides or insecticides are rarely used.

Data on the disease occurrence and associated pathogen species on wheat as well as the agronomic factors influencing diseases are not available in Oman, which is not the case in the UK. This study was conducted to compare the disease incidence of winter wheat in two agro-climatic conditions Oman and the UK between 2009 and 2014. In addition, this study is the first to identify and characterise the pathogenicity of fungal isolates associated with the stem, leaf and ear diseases in Omani wheat and model agronomic factors in disease occurrence. Long runs of data are essential if trends and major changes in

dominance of particular diseases are to be revealed and the probable causes identified. New knowledge will provide guidelines for the formulation of control practices.

In this study, 447 fields in five different locations in Oman were assessed for stem and foliar disease incidence between 2009 and 2013 at flowering growth stage (55-69). In addition, 45 fields were assessed in 2014 at three different growth stages GS 39-51, GS 55-69 and GS 71-87. A questionnaire was designed and detailed information about agronomic practices and disease control was gathered from the growers from all fields sampled and in all years. On the other hand, approximately 300 crops were assessed annually between 2009-2014 in UK, during the early to medium milk development stage (GS73-75). Of them 25 tillers were examined for leaf, stem and ear diseases.

The main aim was to determine the impact of agronomy factors on the occurrence of fungal diseases and severity in Omani wheat. Specifically, the objectives were to i) to compare the incidence of wheat diseases in two different environment Oman and the UK between 2009 and 2014, ii) to identify the main diseases that can potentially cause yield losses in Omani wheat and iii) to characterise the pathogen/s associated with them.

2.2. MATERIALS AND METHODS

2.2.1 COLLECTION OF AGRONOMIC DATA AND SAMPLING

Qualitative and quantitative information on cultivation practices and incidence of foliar diseases was gathered between 2009 and 2013 on 447 fields in five provinces (Figure 2-1), Buraimai, Thahira, Interior, Sharqia and Batainah. Incidence of leaf spot, ear smut, stem and leaf rusts was assessed from winter wheat during the flowering stage (GS 59-69, Zadoks et al., 1974). Fields were crossed in W- shape and 30 plants were assessed across the field. The number of fields selected from each province was different each year based on the proportion to the wheat grown in the province (Table 2-1). During 2014 growing season, 45 wheat fields were sampled by collecting thirty wheat samples at W- traverse of the field at GS 39-51, GS 55-69 and GS 71-87. The incidence of foliar, stem base and ear blight diseases of UK winter wheat were obtained from the CropMonitor (www.cropmonitor.co.uk) disease survey of mainly commercial crops and HGCA Recommended List trials, for up to 30 different winter wheat cultivars. Data was collected as the percentage of sample affected by each disease sampled between 2009 and 2014.

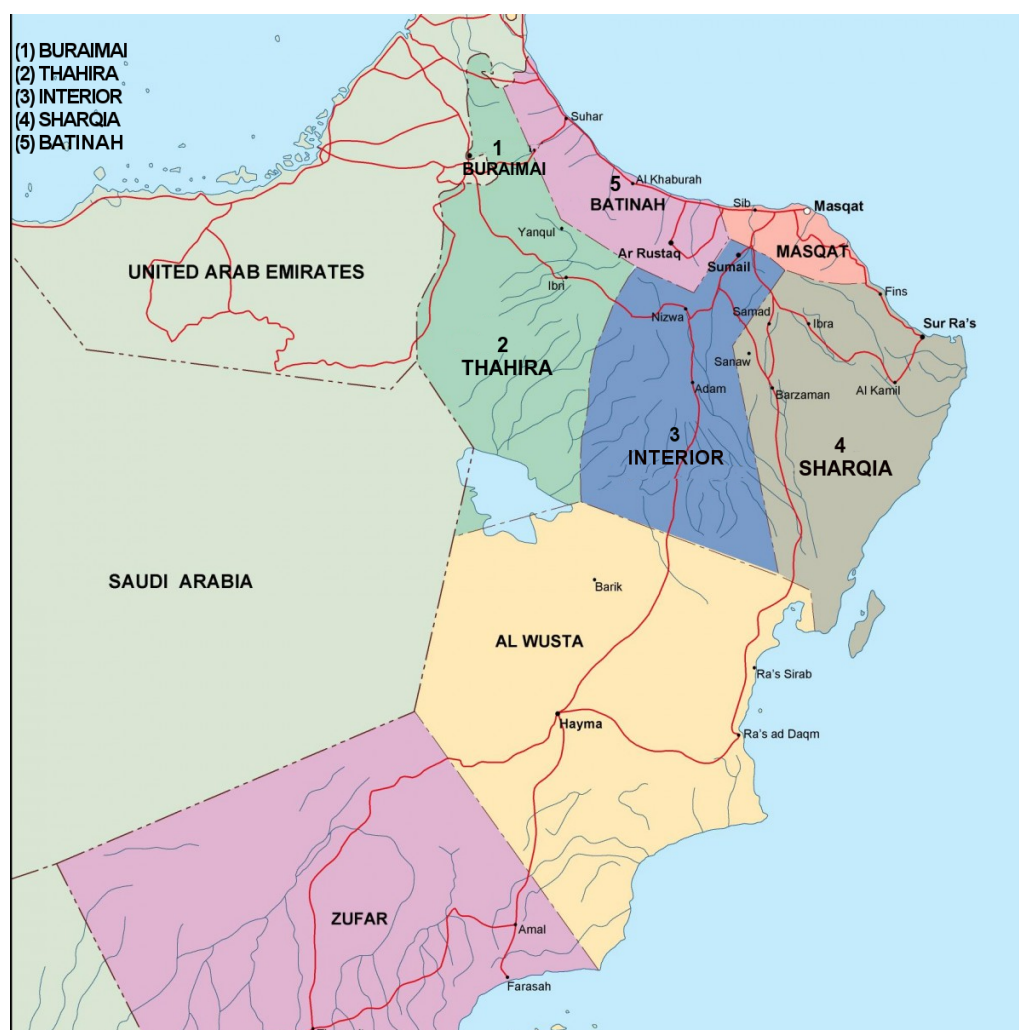


Figure 2-1: Omani provinces with field surveyed indicated by numbers.

Table 2-1: Number of fields and their areas surveyed in Oman between 2009 and 2014.

Year	Provinces	No of fields	Field area
2009	Buraimai	1	< 0.42 ha
		3	>0.5 to < 2 ha
	Thahira	7	< 0.42 ha
		14	>0.5 to < 2 ha
		1	<2.1 to < 4 ha

2010	Interior	4	< 0.42 ha
		14	>0.5 to < 2 ha
	Sharqia	5	< 0.42 ha
		2	>0.5 to < 2 ha
		2	> 2.1 to < 4 ha
	Buraimai	8	< 0.42 ha
		13	>0.5 to < 2 ha
	Thahira	26	< 0.42 ha
		31	>0.5 to < 2 ha
	Interior	4	< 0.42 ha
		8	>0.5 to < 2 ha
		3	> 4.1 to < 6 ha
		1	> 6 ha
	Sharqia	3	< 0.42 ha
		11	>0.5 to < 2 ha
	Batinah	4	< 0.42 ha
		3	>0.5 to < 2 ha
2011	Buraimai	5	< 0.42 ha
		6	>0.5 to < 2 ha
		3	>2.1 to <4 ha
	Thahira	5	< 0.42 ha
		25	>0.5 to < 2 ha
		2	>2.1 to <4 ha
	Interior	3	< 0.42 ha
		7	>0.5 to < 2 ha
	Sharqia	1	< 0.42 ha
		3	>0.5 to < 2 ha
	Batinah	25	< 0.42 ha

		33	>0.5 to < 2 ha
		1	>2.1 to <4 ha
2012	Buraimai	4	<0.42 ha
		4	>0.5 to <2 ha
		5	>2.1 to <4 ha
		1	>4.1 to <6 ha
		1	>6 ha
	Thahira	5	<0.42 ha
		14	>0.5 to < 2 ha
		2	>2.1 to <4 ha
	Interior	3	<0.42 ha
		8	>0.5 to < 2 ha
	Sharqia	1	<0.42 ha
		3	>0.5 to < 2 ha
	Batinah	13	<0.42 ha
		33	>0.5 to < 2 ha
		1	>2.1 to <4 ha
2013	Buraimai	12	< 0.42 ha
		7	>0.5 to < 2 ha
		1	> 6 ha
	Thahira	10	< 0.42 ha
		9	>0.5 to < 2 ha
		1	>2.1 to <4 ha
	Interior	5	< 0.42 ha
		6	>0.5 to < 2 ha
	Sharqia	3	< 0.42 ha
		2	>0.5 to < 2/ha
		1	>6/ha

2014	Batinah	6	< 0.42/ha
	Buraimai	5	<0.42/ha
	Thahira	4	< 0.42 ha
		5	>0.5 to < 2 ha
		1	>2.1 to <4 ha
	Interior	3	< 0.42 ha
		6	>0.5 to < 2 ha
		1	>6/ha
	Sharqia	5	< 0.42 ha
		4	>0.5 to < 2 ha
		1	>6/ha
	Batinah	10	< 0.42ha

Coordinated with visual disease assessment, questionnaire was designed and growers were surveyed about management practices including sowing time, irrigation technique and timing of irrigation, seed source, variety and fertilizer type and application and pesticide use (Table 2-2).

Table 2-2: Management practices and their level gathered during the field survey in Oman.

Management practices	Options
Seed source	Farmer or Ministry
Seed treatment	Yes or NO
Sowing date	Between 15-30 October, between 1-15 November, between 16-30 November, between 1-15 December or >16 December
Tillage	Ploughed or mini-tillage
Sowing method	Manual or mechanical
Previous crop	Fallow, wheat, other cereals or legumes

Variety	Wadi Quriat 226, W.Q. 101, W.Q.308, W.Q. 302, W.Q. 151, W.Q.110 local (Saneen, Missani, Humira, Cooly).
Irrigation system	Flood, sprinkler or drip
Irrigation time	Morning or evening
Use of irrigation /week	1 time, 2-3 times or >4 times
Fertilizer application	Yes or no
Fertilizer type	Manure, urea, NPK, superphosphate or Potassium+ ammonium + foliar
Fertilizer application time	Before sowing, 30 days after sowing or 60 days after sowing
Pesticide application	Yes or no

2.2.2 DISEASE ASSESSMENTS AND IDENTIFICATION

At each GS, collected plants were assessed for leaf, stem-base and ear diseases. Rust and powdery mildew were assessed when symptoms were first observed from GS71-87. Leaf diseases were recorded as percentage area affected on the flag leaf and first leaf of each sample using standard area diagrams (James, 1971). However, stem-base diseases were recorded as percentage of stems with symptoms at the nodes and internodes as well as rot or decay at base that leads to stem weakening, following the method described by Clarkson & Polley (1981). The incidence of disease from 30 samples was calculated as the percentage of stems with visible lesions, where 0 was assigned to symptomless plants. Symptoms were scored as slight (1) when lesions covered less than half of the circumference of the stem; moderate (2) when lesions occupied more than half of the circumference of the stem or severe (3) when the lesions girdled and weakened the stem. Head diseases and loose smut were assessed

once heads emerged GS55-69 using “Horsfall-Barrett” scales that count the infected spikelets and express that as a percentage from the total spikelets of the head. Rusts and powdery mildew at GS71-87 were assessed using the rating scale from James (1971). Severity of rust and powdery mildew were classified into different percentage classes and leaf area affected by disease was recorded.

2.2.3 ISOLATION OF FUNGAL SPECIES FROM SYMPTOMATIC SAMPLES

Symptomatic tissues of stem, leaf or ear were washed using tap water, surface sterilized using 10% sodium hypochlorite (NaOCl), washed in sterile distilled water (SDW) and then blotted dry on sterile filter paper. Three 5-mm pieces of stems, leaves or ears were placed in each Petri-dish containing 2.5% potato dextrose agar (PDA, Oxoid, Hampshire, England). Two Petri dishes were used for each sample, and the plates were maintained at room temperature ($22^{\circ}\text{C} \pm 2$) for 1-3 days. Actively growing mycelia from plant tissues was excised and was sub-cultured into fresh PDA plates. This was followed by producing pure cultures using mycelium tip culture preserved at room temperature in PDA slants amended with 10 mg L^{-1} rifampicin and 100 mg L^{-1} ampicillin to prevent bacterial contamination (Al-Sa’di et al., 2007).

2.2.4 PATHOGEN CHARACTERISATION

DNA was extracted from mycelium using the method described by Al-Sa'di et al. (2007). Freeze dried mycelium was ground, followed by lysis using lysis buffer. Phenol: chloroform: isoamyl alcohol (25:24:1) was added to the mix, followed by centrifugation. Then the DNA was precipitated using NaAc and isopropanol. The DNA pellets were washed with 70% ethanol.

Fungi were identified to the species level based on sequences of the internal transcribed spacer region of the ribosomal DNA (ITS rDNA) as described by Al-Sadi et al. (2011). The universal primers ITS1 (5'-TCCGTAGGTGAACCTGCGG-3') and ITS4 (5'-TCCTCCGCTTATTGATATGC-3') were used to amplify the ITS rDNA region of the fungal isolates (White et al., 1990). The PCR reaction mixture consisted of 0.4 μ M of each primer, 25 ng of DNA samples, PuReTaq™ Ready-To-Go™ PCR beads (GE Healthcare) and Milli-Q water up to a final volume of 25 μ l. Gel electrophoresis was used to check the amplification of the ITS region. The mixture of each reaction of 5 μ l was then run on a 1.5% agarose gel in 0.5x Tris-borate-EDTA buffer (TBE) at 100 V for 60 min.

Samples were sequenced at Macrogen Inc. (Seoul, Korea) using the same primers used for amplification. The alignment and edition of ITS sequences for each isolate was performed using ChromasPro. A BLAST search was then used to compare the representative sequence from each identical set of sequences with worldwide collections of sequences deposited at the National Centre for

Biotechnology Information (NCBI). Phylogenetic analysis of the sequence data was done by comparing sequences obtained in this study with sequences of reference isolates from GenBank. Trees were constructed based on pairwise distances obtained using the Kimura 2 parameter evolutionary model (Mega 5) (Tamura et al., 2013).

2.2.5 PATHOGENICITY TEST

Pathogenicity tests were conducted for the four most common fungal species (*Alternaria alternata*, *Bipolaris sorokiniana*, *Setosphaeria rostrata*, *Fusarium equiseti*), associated with foliar diseases of wheat on two wheat cultivars, Cooly and WQ226. Wheat seeds were sown in 15-cm pots, 20 seeds per pot. Spore suspension was prepared for each fungal species and adjusted to 100 spore's μl^{-1} . Conidia were applied on the leaves of 2-weeks old wheat seedlings. Wheat seedlings were irrigated daily using 50 ml of water and covered with polyethylene bag for 24 hr. Three replicate pots were used for each fungal species-wheat cultivar combination and the pots were kept at 24°C for 14 days after inoculation (dai). Severity of the disease was recorded as percentage of the leaf area covered with chlorosis/necrosis as described by Al-Sadi (2015). Re-isolations were made from leaves developing leaf spot symptoms to confirm Koch's postulates. Data from pathogenicity tests were analysed using Tukey's Studentized range test, using Statistical Analysis Software (SAS Institute Inc., NC, and USA).

2.3. RESULTS

2.3.1 DISEASE INCIDENCE AND SEVERITY BETWEEN 2009 AND 2014 IN OMAN AND UK

The survey was conducted between 2009 and 2014; during this period 492 fields were examined at growth stages 55-69 for diseases on wheat grown in five provinces in Oman (Figure 2-1). The disease incidence data for the UK winter wheat were collated from the CropMonitor disease survey of mainly commercial crops and HGCA Recommended List trials, for up to 30 different winter wheat cultivars (www.cropmonitor.co.uk, Figure2-3). These data were recorded as percentage plants affected by different diseases from 300 crops, which were sampled during July/August between 2009 and 2014 as part of annual surveys.

The disease incidence of leaf spot in Omani wheat increased from 18% in 2009 to 42.2% in 2013 and was the predominant disease in wheat crops between 2010 and 2013. Loose smut and stem-base diseases predominated at 22% and 45%, in 2009 and 2014, respectively. The incidence of powdery mildew was highest in 2009 (7%) and lowest in 2012 (1%), (Figure 2-2).

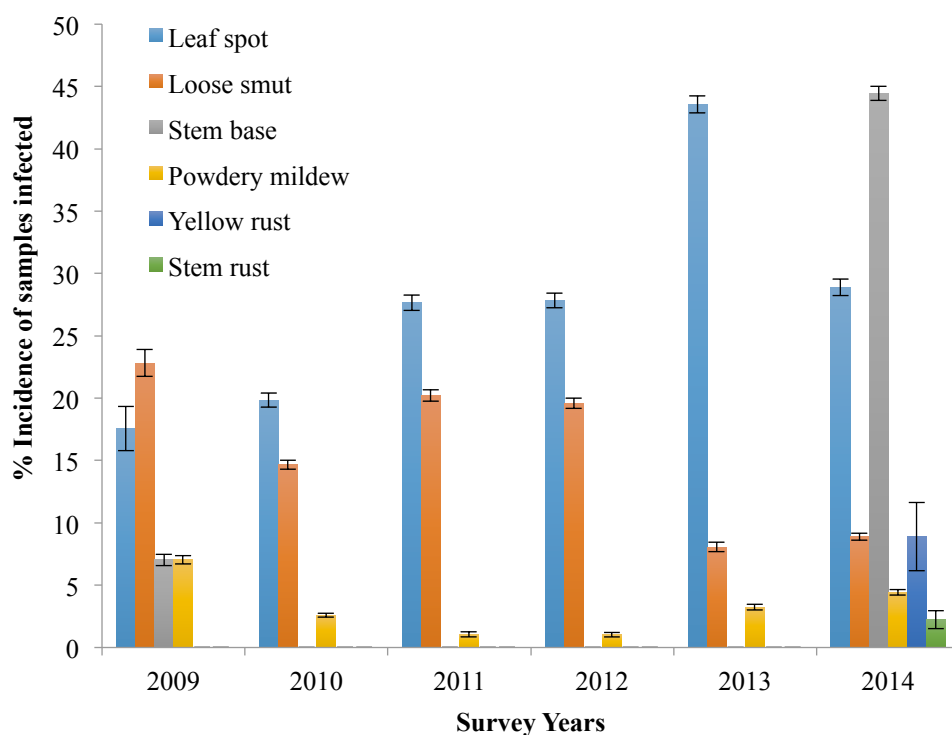


Figure 2-2: Incidence of disease in Oman wheat field assessed at GS55-69 between 2009 and 2014 Bars=1±SEM, assessed by multiple regression. Leaf spot disease increased from 17% incidence of samples in 2009 to 44% incidence in 2013. Stem base diseases were recorded only in 2009 and 2014 with highest incidence in 2014 more than any other disease. Stem and yellow rust were recorded only in 2014 with less than 10% incidences. Loose smut was common in all years with more than 15% of the samples in the early years of the survey then decreased in 2013 and 2014 to less than 9% of the samples. Powdery mildew was recorded in less than 8% incidence of the samples in all years of the survey.

Significant Stem base ($P=0.005$), Loose smut ($P<0.001$), Yellow rust ($P<0.001$).

The predominant disease in the UK winter wheat between 2009 and 2014 was Septoria disease and it was the most common foliar disease with 99% incidence recorded in 2014. The disease incidence of tan spot ranged between 6% and 20% of samples infected with the highest incidence recorded in 2011, (Figure 2-3). The incidence of stem-base diseases was lower than 40% of samples infected in all years with the highest incidence (37%) recorded in 2013. Fusarium was most common followed by eyespot diseases. Powdery mildew was highest in 2009 (36% incidence) and lowest in 2012 (4% incidence), (Figure 2-3).

Ear blight diseases were recorded the highest in samples infected in 2012 with incidence of 96%, up from 21% recorded in 2011, which was the lowest incidence among the 6 years surveyed. Yellow rust was recorded in less than 10% of plant sampled in all years, whilst highest record of brown rust was in 2012 with 17% of plant samples and lowest in 2010 with no samples infected with this disease. Glume spot disease was recorded highest in 2009 with 65% of samples infected while in all following year the samples infected with glume spot was less than 60% with lowest recorded in 2013 (27%), (Figure 2-3).

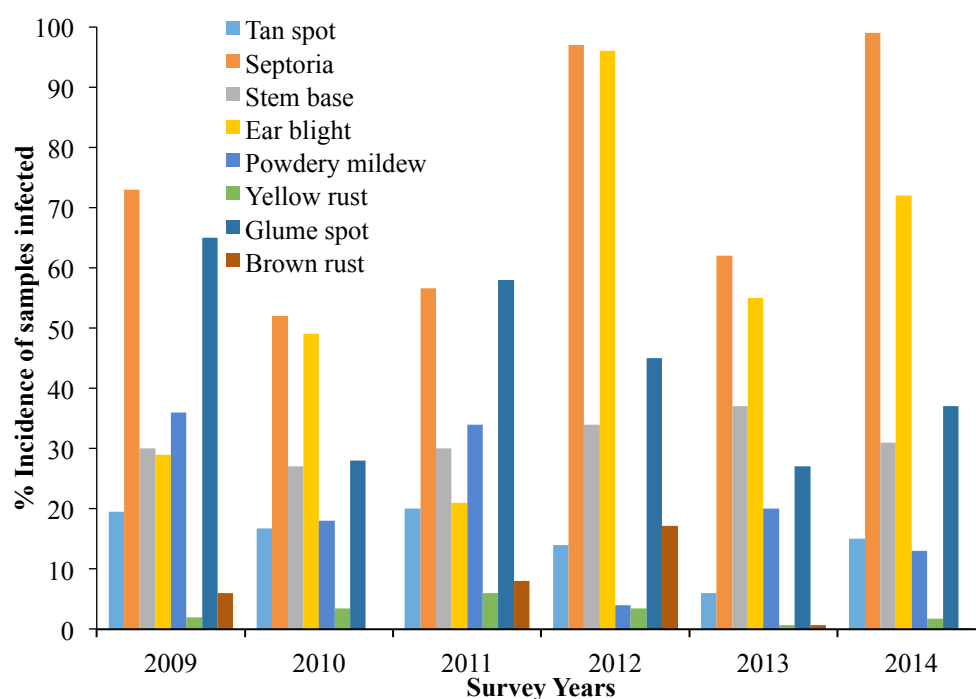


Figure 2-3: Incidence of disease in the UK wheat field assessed between 2009 and 2014. *Septoria* was common foliar diseases in all surveyed years and reached its highest incidence in 2014 with 99% of the samples. Fusarium was the common disease of the stem base ranging from 27% to 37% incidence between 2009 and 2014. Eyespot was the second most important of the stem base diseases in all surveyed years. Brown rust fluctuated through surveyed years with highest record of 17% incidence of sampled crop and no record in 2014. Ear blight recorded in high incidence in most years reached its maximum in 2012 with 96% incidence. Glume spot was ranging from 65% to 27% incidence during the surveyed years. Tan spot disease was recorded in less than 20% and powdery mildew recorded in less than 36 % incidence of the crop sampled in all years. The incidence of Yellow rust was low in all years with 6% as the highest in 2011 and 1% as the lowest in 2013.

Leaf spot influenced by sowing method, location (provinces) and variety, Table 2-3. Shariqia province had higher incidence of disease compared to Batinah ($p < 0.001$). Mechanical sowing method was predicted to increase

leaf spot more than manual method. In addition W.Q. 302 variety was associated with higher incidence of leaf spot, while W.Q. 101 had least disease.

Stem-base disease was influenced by year, sowing method and irrigation, with year being highly significant ($p < 0.001$), (Table 2-3). Drip irrigation was associated with highest stem base disease than other methods. Incidence was highest in 2014. Mechanical sowing increased stem-base more than manual method. Loose smut disease was influenced by year, sowing method, location and variety. W.Q 110 was the variety associated with high incidence of loose smut while the lowest incidence was predicted on W.Q. 151. Loose smut disease was favoured by mechanical sowing much more than by manual sowing, (Table 2-4).

Table 2-3: Multiple regression models on incidence (%) of Leaf Spot, Stem base, loose smut and yellow rust diseases assessed at GS55-69 in Omani wheat fields 2009-2014, No. of fields =468, Total d.f.=467

Fixed Term	Leaf Spot			Stem base			Loose Smut			Yellow rust		
	s.s	d.f.	p	s.s	d.f.	p	s.s	d.f.	p	s.s	d.f.	P
Year	-	-	-	293.3	5	<.001	250.5	5	0.014	1095.3	5	<.001
Sowing method	296.4	1	0.010	20.3	1	0.005	605.7	1	<.001	623.2	4	<.001
Provinces	1978.15		<.001	-	-	-	586.4	5	<.001	552.8	4	0.004
Variety	1196	9	0.002	-	-	-	646	9	<.001	-	-	-
Irrigation	-	-	-	34	2	0.001	-	-	-	-	-	-

*d.f. =Degree of freedom, S.S= Sum of square.

Leaf spot disease was influenced significantly by sowing method, location and variety. Stem base diseases were influenced significantly by year, sowing method and irrigation. Loose smut was influenced by year, sowing method, location and variety. Yellow rust was influenced by year, sowing method and location.

Table 2-4: Predictions of incidence (%) from multiple regression models of leaf spot, loose smut, yellow rust and stem base diseases assessed at GS55-69 in Omani wheat fields 2009-2014, No. Of fields =468, Total d.f=467.

Factor	Predicted incidence (%)			
	Leaf spot	Loose smut	Yellow rust	Stem base
Years				
2009		2.80	0.00	0.83
2010		1.13	0.01	0.00
2011		2.44	0.05	0.04
2012		2.40	0.09	0.04
2013		1.40	0.14	0.00
2014		0.35	4.85	2.67
SED average		0.75	1.02	0.27
Provinces				
Buraimai	2.89	2.03	0.29	
Thahira	1.30	0.97	0.00	
Interior	5.94	2.53	2.68	
Sharqia	6.76	3.81	0.00	
Batinah	1.26	1.17	0.20	
SED average	1.71	1.07	1.36	
Sowing method				
Manual	2.96	1.63		0.31
Mechanical	6.85	6.31		1.61
SED average	1.65	1.03		0.39
Variety				
Wadi Quriat 226	3.62	1.01		
Wadi Quriat 308	2.15	1.30		
Wadi Quriat 110	3.49	3.43		

Cooley	2.25	0.99
Saneen	2.69	0.71
Missani	2.05	1.95
Wadi Quriat 302	15.93	3.31
Wadi Quriat 101	1.17	2.38
Humira	4.52	0.98
Wadi Quriat 151	1.74	0.70
SED average	2.31	1.45
<hr/>		
Irrigation system		
Flood		0.34
Sprinkler		0.32
Drip		1.39
SED average		0.37
<hr/>		
Sowing date		
Between 15-30 October		0.35
Between 1-15 November		0.32
Between 16-30 November		0.25
Between 1-15 December		5.04
>16 December		0.00
SED average		1.72

***SED= Average standard error of difference**

The incidences of loose smut ranged from 2.8% in 2009 to 0.35 in 2014, while yellow rust ranged from 0% in 2009 to 4.9% in 2014. The highest incidence of stem base disease was 2.7% in 2014 and lowest was 0% in 2010 and 2013. The highest incidence of leaf spot was located in Sharqia with 6.8% and lowest incidences located in Batinah with 1.2%. Incidence of loose smut was highest in Sharqia with 3.8% and lowest in Thahira with 1% incidence. Yellow rust presence was high in Interior with 2.8% and low in Thahira with 0%. Mechanical sowing method resulted in high incidences in all diseases. The highest presence of Leaf spot was in Wadi Quriat 302 variety with 16% incidence and lowest in Wadi Quriat 101 with 1.2% incidence. Presence of Loose smut incidence was high in Wadi Quriat 110 and low in Wadi Quriat 151. Stem base disease was present with high incidence in drip irrigation method, while Yellow rust was present high in sowing dates between 1 and 15 December.

2.3.2 DISEASE INCIDENCE AND SEVERITY IN 2014

Forty-five fields were examined at three different stages (GS39-51, 55-69 & 71-87) in Buramiai, Thahira, Interior, Batinah and Sharqiah provinces. The majority of the 45 wheat fields that were surveyed in 2014 were found to exhibit symptoms of stem rot and leaf spots and browning and spotting of the ears. Leaf spot incidence decreased as the crop matured from 42.2% at GS 39-51 to 28.89% at GS71-87, (Figure 5-4). Stem base disease incidence increased throughout crop development reaching 46.67% at GS71-87. Ear disease was 42.2% at GS55-69 but symptoms were less visible and decreased by two-fold as the crop matured by GS71-87. Loose smut was recorded at 8.89% at GS71-87 and increased by two fold at GS55-69. Stem rust and powdery mildew at GS71-87 occurred in 2.2% and 4.4% of fields, respectively.

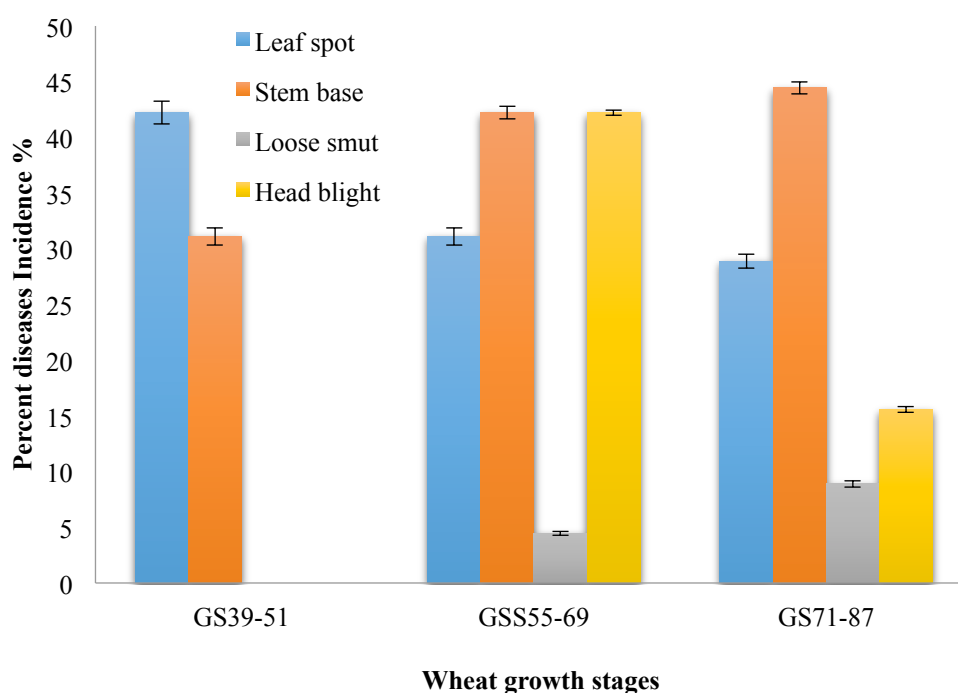


Figure 2-4: Incidence of diseases in Omani wheat crop assessed at GS39-51, GS55-69, and GS71-87. Bars= $1\pm\text{SEM}$, analysed by residual maximum likelihood (REML). Leaf spot disease decreased as the crop matured from 42% incidence at GS39-51 to 28% at GS71-87. Stem base disease increased as the crop matured reaching 44% incidence at GS71-87 from 31% incidence at GS39-51. Head blight was recorded high at flowering stage with 42% but decreased to 15% incidence at GS71-87. Incidence of Loose Smut increased from 4% at GS55-69 to 8% at GS 71-87. Significant Leaf spot ($p=0.028$), Loose smut ($p=0.004$).

2.3.3 FUNGAL PATHOGENS ASSOCIATED WITH WHEAT

Isolations from 45 fields growing 6 different wheat varieties resulted in 36 fungal species isolations. *Alternaria alternata* was the most dominant pathogen isolated with 18% frequency. The common pathogens from each tissue can be found in Table 2.5. With *A. alternata*, *Bipolaris sorokiniana* and *Setosphaeria rostrata* are the most common species recovered from

leaf, stem and ears. *A. alternata* was recovered from (6) wheat varieties and provinces surveyed. However, *B. sorokiniana* was recovered from (3 varieties, all provinces), *S. rostrata* (4 varieties, all provinces). The other 33 fungal species identified from this study varied in frequency from 0.6 to 7.3%, (Table 2-5).

Table 2-5: Geographical distribution, growth stage (GS), tissues and wheat hosts “variety” of fungi recovered from stem-base, leaf and ears of wheat in Oman 2013/2014.

Fungal species	% Recovery	(GS) recovery	Provinces	Recovery tissues	Variety
<i>(Alternaria alternate)</i>	18	All	All	All	All
<i>(Bipolaris sorokiniana)</i>	11.3	All	All	All	1,2,3
<i>(Setosphaeria rostrata)</i>	10.6	All	All	All	1,2,3,5
<i>(Fusarium equiseti)</i>	7.3	All	1,2,3,4	All	1,2,3,4
<i>(Alternaria tenuissima)</i>	6.6	All	All	L&S	2,4,3,6
<i>(Alternaria infectoria)</i>	6	All	1,3,4	L&S	1,2,3,4
<i>(Marasmius nigrobrunneus)</i>	4.6	All	1,3,4	All	1,2,4
<i>(Alternaria brassicicola)</i>	4	All	1,4,5	All	1,2,3
<i>(Cladosporium cladosporioides)</i>	2.6	55-69/71-87	3	L&S	1,2,4
<i>(Stemphylium globuliferum)</i>	2.6	39-51/71-87	1,4	L&S	1,6
<i>(Puccinia triticina)</i>	2	71-87	3,5	L	2,4,5
<i>(Stemphylium)</i>	2	55-69/71-	1,3,4	L&S	2,4

<i>vesicarium</i>)		87			
<i>Rhizopus oryzae</i>	2	39-51	1,4	S	1,2
(<i>Fusarium</i>	1.3	55-69/71-	2,3	S	1,3
<i>nygamai</i>)		87			
<i>Pleospora allii</i>	1.3	71-87	2	L	4
(<i>Curvularia</i>	1.3	71-87	3,5	S	1,5
<i>australiensis</i>)					
(<i>Alternaria</i>	1.3	39-51/55-	3,4	L&E	2,4
<i>arborescens</i>)		69			
(<i>Bipolaris</i>	1.3	55-69	3,4	L	2,4
<i>tetramera</i>)					
(<i>Fusarium</i>	1.3	39-51/55-	1,2	S	1
<i>oxysporum f. sp</i>)		69			
(<i>Blumeria</i>	0.6	71-87	1	L	1
<i>graminis</i>)					
(<i>Fusarium</i>	0.6	39-51	1	S	6
<i>nectrioides</i>)					
(<i>Nigrospora</i>	0.6	39-51	1,5	S	3
<i>sphaerica</i>)					
(<i>Mucor</i>	0.6	39-51	4	L	2
<i>circinelloides</i>)					
<i>Rhizoctonia sp</i>	0.6	55-69	3	L	4
(<i>Cochliobolus</i>	0.6	71-87	1	S	1
<i>australiensis</i>)					
(<i>Epicoccum</i>	0.6	55-69	2	S	1
<i>nigrum</i>)					
(<i>Gibberella</i>	0.6	55-69	3	S	3
<i>moniliformis</i>)					
(<i>Fusarium</i>	0.6	71-87	4	S	2
<i>incarnatum</i>)					
(<i>Pythium rhizo-</i>	0.6	71-87	5	L	5
<i>oryzae</i>)					
(<i>Fusarium</i>	0.6	71-87	5	L	3
<i>acutatum</i>)					
(<i>Pleospora</i>	0.6	71-87	3	L	4
<i>herbarum</i>)					

<i>Alternaria citri</i>	0.6	71-87	3	L	4
<i>(Epacris microphylla)</i>	0.6	71-87	2	S	2
<i>(Alternaria solani)</i>	0.6	71-87	2	S	3
<i>(Puccinia graminis f. sp. tritici)</i>	0.6	71-87	1	S	1

***(GS)= growth stages (39-51/55-69/71-87), *variety: 1=W.Q.226, 2=W.Q.308, 3=W.Q.310, 4=Cooley, 5=Saneen, 6= Humira. *Provinces: 1=Buraimai, 2=Dhairah,3=Dkiah,4=Batinah,5=Sharqiah.,*Recovery Tissues= L=leaf, S=Stem, E=Ear**

All species were identified by culturing then sequencing except yellow leaf rust (*Puccinia triticina*), Stem rust (*Puccinia graminis f. sp. tritici*) and powdery mildew (*Blumeria graminis*) were sequenced directly.

***Alternaria alternata* was the most dominant pathogen isolated from all tissues with 18% frequency followed by *Bipolaris sorokiniana* and *Setosphaeria rostrata*. The other 33 fungal species isolated varied in frequency from 7.3 to 0.6%.**

The main factors influencing leaf spot development in 2014 were location (provinces) ($P= 0.005$) and application time of urea fertilizer ($P= 0.013$), (Table 2-6). Highest leaf spot incidence was in Buramai province with the mean of 1.33%, however leaf spot incidence in Sharqia was the lowest with 0.35%. The application of urea fertilizer after 2 months from sowing was found to be the lowest to influence leaf spot incidence (0.29%); whilst urea application one month after sowing cause highest leaf spot incidence (1.2%). However, applying urea before sowing influencing leaf spot incidence with 0.53%, (Table 2-7).

Stem base diseases influenced by locations ($P= 0.010$) and urea fertilizer application ($P= 0.018$). The highest incidence of stem base diseases was obtained from Thahira province (1.62%), while Batinah had lowest stem base incidence (0.37%). The result revealed that fields fertilized with urea

had highest stem base incidence (1.28%), comparing to those fields without urea application (0.71%), (Table 2-7). Loose smut disease was influenced by application of potassium + ammonium foliar fertilizer only ($P= 0.021$), (Table 2-6).

Table 2-6: Mixed model restricted maximum likelihood (REML) analysis on disease index of leaf spot, stem base & loose smut assessed at GS25-51, GS55-69 & GS71-87 in Omani wheat fields 2014, No. of fields =45, Total d.f=44.

Fixed Term	Leaf Spot			Stem base			Loose smut		
	WS	d.f.	p	WS	d.f.	p	WS	d.f.	p
Provinces	17.37	4	0.005	15.31	4	0.010	-	-	ns
Urea application Time	9.78	2	0.013	-	-	ns	-	-	ns
Foliar application rate of potassium + ammonium	-	-	ns	-	-	ns	5.76	1	0.021
Urea application	-	-	ns	5.13	1	0.018	-	-	ns

ns= Not Significant

Leaf spot diseases were influenced significantly by location and time of urea application. Stem base diseases were influenced significantly by location and urea application. Loose smut was influenced significantly by the rate of potassium and ammonium application.

Table 2-7: Predictions of incidence (%) analysed by residual maximum likelihood (REML) of leaf spot, stem base & loose smut assessed at GS25-51, GS55-69 & GS71-87 in Omani wheat fields 2014, No. of fields =45, Total d.f=44.

Factor	Predicted incidence (%)		
	Leaf spot	Stem base	Loose smut
Provinces			
Buraimai	1.33	0.72	
Thahira	0.62	1.62	
Interior	0.68	1.38	

Sharqia	0.35	0.90
Batinah	0.38	0.37
SED average	0.24	0.41
Urea application Time		
Before sowing	0.53	
30 days after sowing	1.19	
60 days after sowing	0.29	
SED average	0.25	
Foliar application rate of potassium + ammonium		
1-10 grams		1.48
No application		0.26
SED average		0.56
Urea application		
NO	0.71	
Yes	1.28	
SED average	0.35	

*SED= Average standard error of difference

The highest incidence of leaf spot was located in Buraimai with 1.3% and lowest incidences located in Sharqia with 0.35%. Incidence of stem base was highest in Thahira with 1.6% and lowest in Batinah with 0.37% incidence. The application of urea after 30 days from sowing predicated high incidence among other application time reaching to 1.2%. The application rate 1-10 gram of foliar potassium and ammonium predicated high incidence of loose smut compared to no application. Urea application increased the chance of stem base disease by 1.3%.

2.3.4 PHYLOGENETIC ANALYSIS

Contiguous sequences were successfully derived from the isolates. The ITS rDNA regions of *B. sorokiniana*, *A. alternata* and *F. equiseti* isolated were further amplified and sequenced. Analysis of the three isolates revealed high

nucleotide similarity to the other sequences from the Genbank. The isolate *B. sorokiniana* showed high nucleotide similarity (99.8%) to isolates (AF158104 and AF158105; Figure 2-5). The Omani isolates also clustered with other isolates of *B. sorokiniana* that have been separated from *B. heterosptrophus* and *B. victoriae* with a very high bootstrap support value (99 and 95% respectively). Omani isolates of *A. alternata* showed (99.8%) nucleotide similarity to other sequences of *A. alternata* from GenBank as shown in Figure 2.6. There is a 100% identical bootstrap of Omani isolate *Fusarium equiseti* with the isolate (GU934522) from the GenBank as Figure.2-7 showed. *F. equiseti* and *F. brachygibbosum* sharing same clade though *F. solani* has different ancestor.

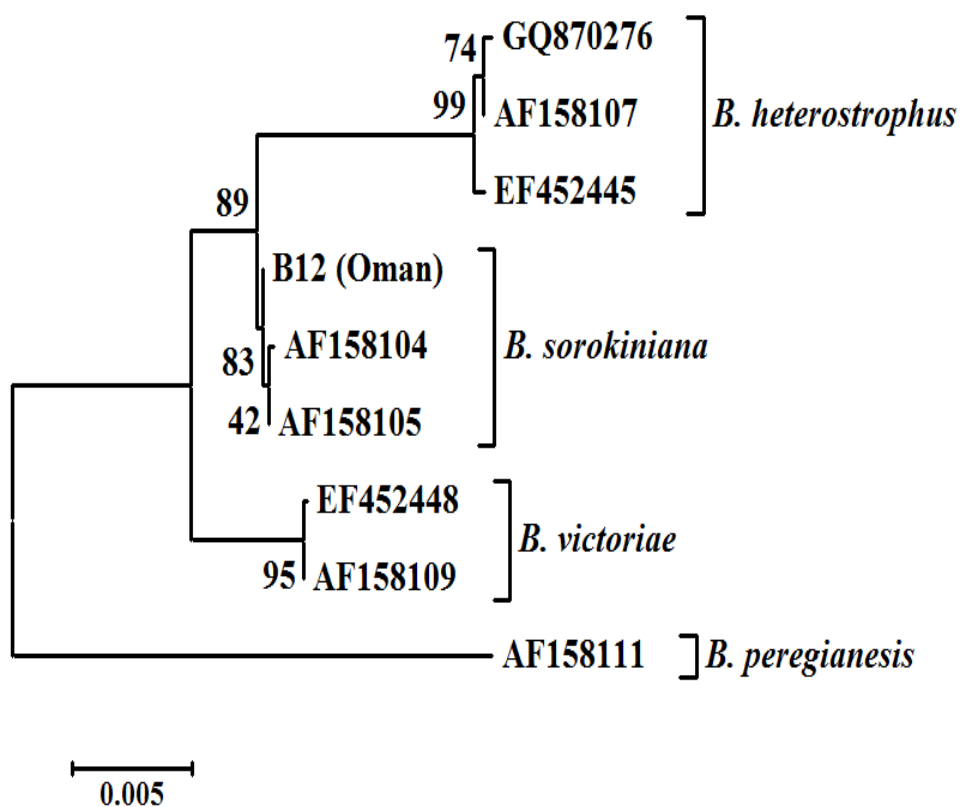


Figure 2-5: A phylogram showing the relationship of *B. sorokiniana* to *B. sorokiniana* from GenBank and to three other *Bipolaris* species based on the ITS rDNA sequences. Bootstrap values are displayed in nodes (1000 replications). The tree is rooted to *B. peregianensis* (AF158111).

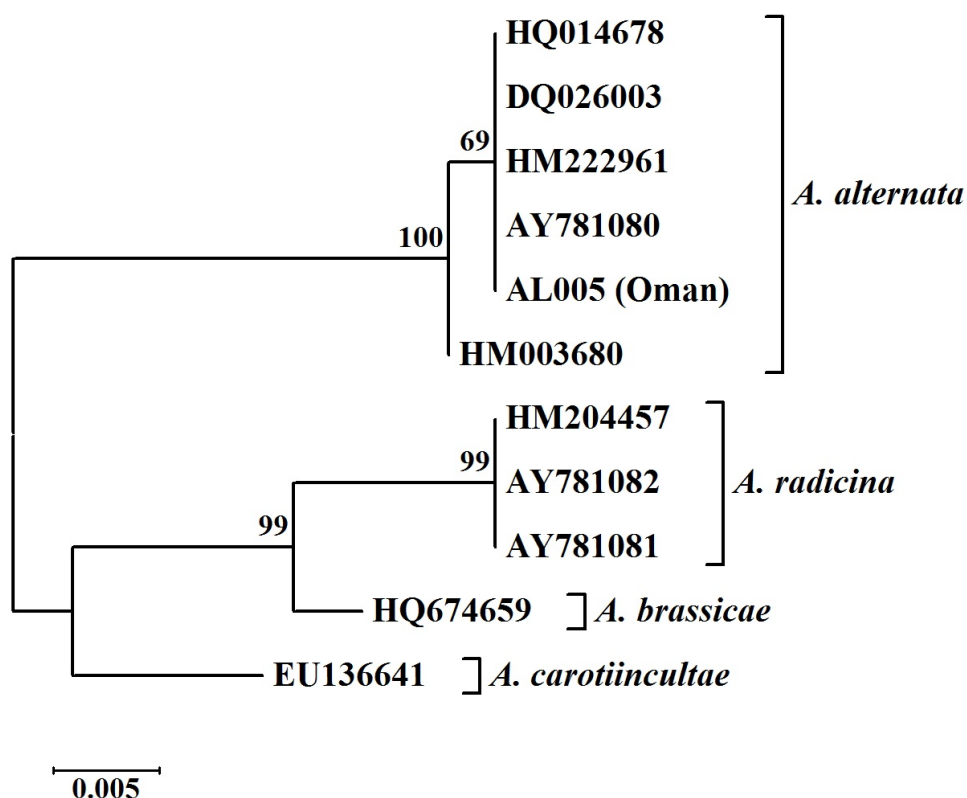


Figure 2-6: A phylogram showing the relationship of *Alternaria alternata* to other isolates and species of *Alternaria* from GenBank based on the ITS rDNA sequences. Bootstrap values are displayed in nodes (1000 replications).

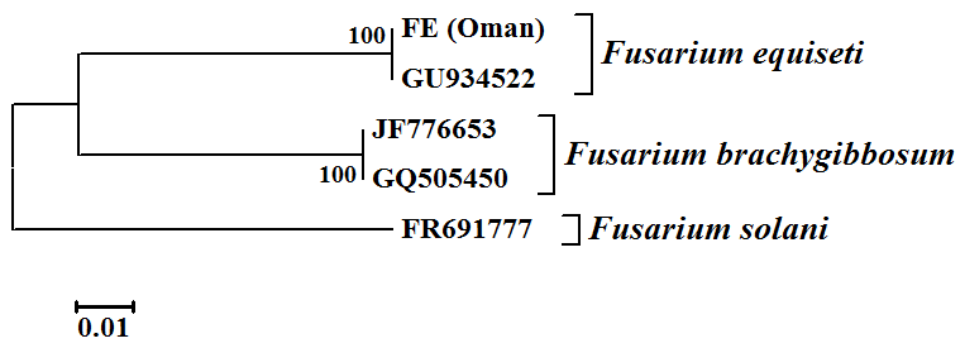


Figure 2-7: A phylogram showing the relationship of *Fusarium equiseti* to other isolates and species of *Fusarium* based on the ITS rDNA sequences. Bootstrap values are displayed in nodes (1000 replications).

2.3.5 PATHOGENICITY

Two varieties were used to confirm that all isolates are pathogenic on both varieties, regardless of whether their resistance is similar. Analysis showed that all isolates are pathogenic on both varieties, but it is very likely that the two varieties have the same level of resistance to all isolates. The inoculated fungi resulted in varying degrees of chlorosis and necrosis on the two wheat cultivars however there were no significant differences between the combinations. *Bipolaris sorokiniana* caused 65.63% chlorosis on Cooly variety of wheat but on WQ226 variety *Alternaria alternata* caused 51.25% of chlorosis. However, *Fusarium equiseti* caused smaller chlorotic lesions in both varieties Cooly and W.Q.226 with 28% and 19% respectively, compared to *Bipolaris* and *Alternaria*. chlorotic lesions, (Figure 2-8). Necrotic lesions were smaller than chlorotic lesions. *B. sorokiniana* caused more necrosis (1.88%) in Cooly compared to the necrosis caused by W.Q.226 cultivar (1.25%). The other pathogens caused less or no necrotic lesions, (Figure 2-9).

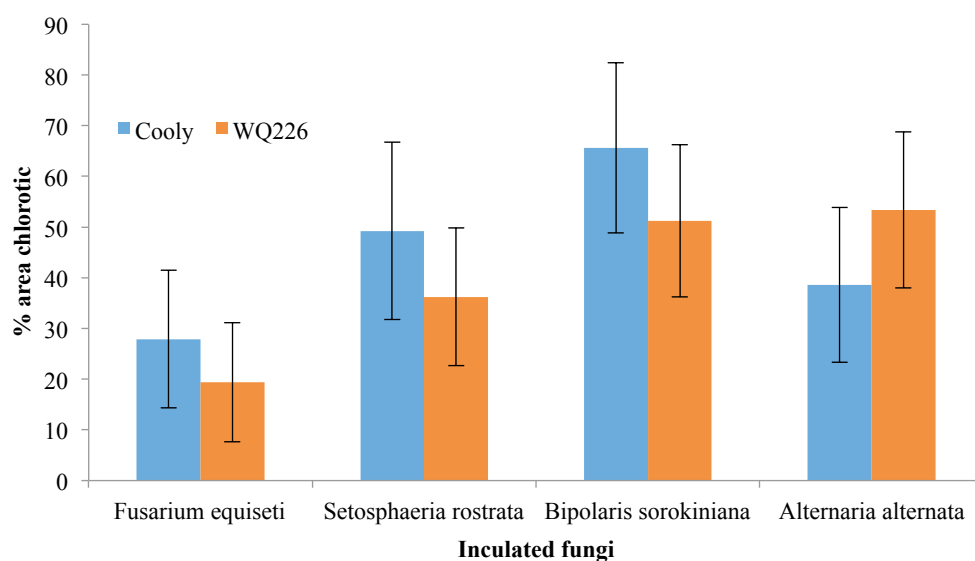


Figure 2-8: Percentage of the wheat leaf area covered with chlorotic symptom as a result of inoculated fungi in two different wheat varieties in Oman Bars=1±SEM. No significant difference was observed in the reaction of wheat varieties to fungal pathogens ($P > 0.05$; Tukey's Studentized range test, SAS).

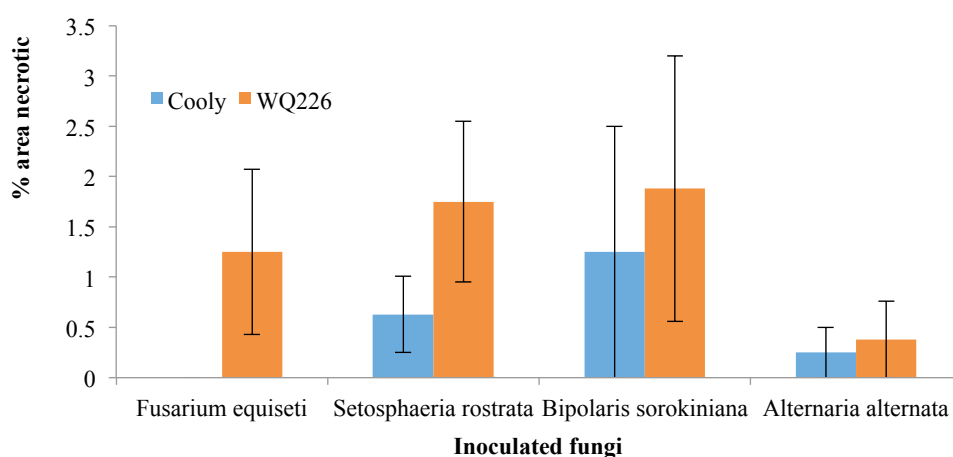


Figure 2-9: Percentage of the wheat leaf area covered with necrotic symptom as a result of inoculated fungi in two different wheat varieties in Oman Bars=1±SEM. No significant difference was observed in the reaction of wheat varieties to fungal pathogens ($P > 0.05$; Tukey's Studentized range test, SAS), except when *F. equiseti* was used.

2.4. DISCUSSION

The incidence of wheat diseases in two different environments, Oman and the UK, were compared between 2009 and 2014. At flowering growth stage (GS 55-69), 447 fields in five different locations were assessed in Oman for stem and foliar disease incidence between 2009 and 2014. Most of the fields surveyed were found to have at least one disease. Brown to black lesions or spot symptoms were observed on the stem, leaf and ears of the plants with some rotting in the affected stem. On the other hand, approximately 300 crops were assessed annually between 2009-2014 in the UK, during the early to medium milk development stage (GS73-75). Of them 25 tillers were examined for leaf, stem and ear diseases.

The incidence of diseases in Omani wheat assessed at growth stage (GS55-69) only between 2009 and 2014 varied. Leaf spot incidence increased through the survey years from only 18% in 2009 to 42.2% recorded in 2013 followed by a decrease to 28% in 2014. However, stem base incidence was lowest in 2009 at 7% and highest in 2014 when it was the predominant disease with 44%. Loose smut incidence fluctuated through the years of the survey between 22% and 8%. Powdery mildew was also recorded through the years of the survey but at lower incidence in all years compared to other diseases. During the more extensive 2014 survey, the peak of leaf spot disease incidence among other diseases was recorded at GS31-59. The most prevalent disease was leaf spot followed by loose smut. It seems also that leaf spot represents the most important disease as its incidence increased

through years and it was recorded with high frequency among other diseases. This finding is supported by reports from different parts of the world that found leaf spot disease infecting wheat at early growth stages (Reamaekers, 1988; Reis, 1991; Schilder & Bergstrom, 1993; Mehta, 1996; Shazia & Iftikhar, 2005; Hajihasani et al., 2012).

In contrast, *Septoria* disease in UK winter wheat was more widespread than any other foliar disease in all years except 2011. It showed wide year-to-year variation, with the highest incidence was present in 99% of crops in 2014. Powdery mildew levels were the second highest foliar disease in all years except 2012 and 2014 followed by tan spot diseases. The incidences of powdery mildew ranged between 36% and 4%, while tan spot incidences ranged between 20% and 6% of samples infected. Of the stem-base diseases, there was little fluctuation between 2009 and 2014 in the percentage of crop affected. Fusarium was generally more common than eyespot and sharp spot diseases with eyespot always more common in all years than sharp spot. The highest incidence of fusarium diseases were recorded in 2013 with 37% while the lowest level was 24% recorded in 2009.

Eyespot severity was highest in 2012, when moderate or severe lesions affected 7-3% of the stems. While in 2014 severity were 6-2% of stems affected by moderate or severe lesions. It is possible that the survey tended to underestimate the severity of eyespot. This may be related to the difficulty to assess the lesions at GS73-75 or lesions being subjected to fungicide treatments. The incidence of sharp eyespot was always lower than that of

eyespot and less severe in all years. The severity of ear blight fluctuated among surveyed years with highest incidence recorded in 2012. The incidence of yellow rust did not exceed 10% of sampled crop in all years and this may be due at GS75 lesions being treated by fungicides or not visible. On the other hand, the severity of brown rust apart from moderate levels in 2012 did not affect more than 8% in all other years with none recorded in 2014. However, glume spot showed wide year-to-year variations with highest severity recorded in 2009 then decreased in all other years to less than 60% of crop infected.

The data presented here records the changes that occurred in the disease of winter wheat in a 6-year period from 2009 to 2014 in two different climates, Oman and the UK. In this six-years period *Septoria* and powdery mildew were the most severe of the foliar diseases in the UK, whereas, leaf spot was the most severe of the foliar diseases in Oman. On the other hand, *Fusarium* and eyespot were the most severe of the stem base diseases in the UK. However, *Fusarium* was the most severe of the stem base diseases in Oman; this may be due to the introduction of sprinkler irrigation. Clearly there is a difference in disease existence and severity and this may be related to the differences in climatic conditions and environments in Oman and the UK. Such surveys provide continuing insight into how the agricultural community responds to change and how this impacts on disease incidence and severity. This assists in developing an understanding of how diseases can be managed in a more rational and sustainable way.

The main diseases causing yield loss in Omani wheat were identified and pathogen associated with them were characterised. To our knowledge this is the first survey on the occurrence of fungal diseases and characterise the pathogens associated with stem, leaf and ear tissues and the influence of agronomy factors. Five provinces (Buraimai, Thahira, Interior, Sharqia and Batinah) were chosen because of their more intensive wheat growing area in Oman. Large numbers of pathogenic fungi causing symptoms were found to be prevalent in wheat fields in Oman.

Isolation from six symptomatic wheat varieties resulted in 36 different fungal species. *Alternaria alternata* was the most frequently isolated pathogen followed by *Bipolaris sorokiniana*, *Setosphaeria rostrata*, and *Fusarium equiseti*. *Alternaria* is a leaf blight fungus commonly isolated from the infected tissues. Also *B. sorokiniana* and *A. alternata* were the most virulent in the pathogenicity tests. *A. alternata* is considered to be an important pathogen in wheat; it was more prevalent compared to other fungi isolated in this study. Joshi et al. (1986) reported that occurrence of blight disease in Pakistan was caused by *A. alternata* that usually isolated from the leaves of the crop during heading stage. In India, this disease represents one of the threatening plant diseases and can cause considerable losses in wheat crop estimated around 10-25% (Singh & Srivastva, 1997).

A survey to investigate the prevalence of foliar blight of wheat rotated with rice in Punjab revealed that the number of foliar samples with *Pyrenophora tritici-repentis* was less as compared with *A. alternata*, *Stemphylium* sp., and

B. sorokiniana (Shazia & Iftikhar 2005). In another study Maraite et al. (1998) found that *B. sorokiniana* was associated with 81% of analysed foliar samples from infected wheat grown in hot and humid areas. Elsewhere in the Golden triangle of Montana, Canada and USA, *B. sorokiniana* was the most widespread crown pathogen of wheat (Moya-Elizondo et al., 2011). The findings from this survey in Omani wheat are similar to that found in Pakistan and India and the USA in respect to *A. alternata* and *B. sorokiniana*. In Oman, these fungi have been isolated from seeds of wheat and only *B. sorokiniana* was found to cause root and crown rot in wheat (Al-Sadi & Deadman, 2010). Both pathogens were recovered from infected stems and leaves in all growth stages as well as from all provinces where temperature and humidity at the time of the survey is suitable for the fungus to grow.

Among the pathogens that have been recovered with high frequency from all growth stages and all locations covered by this survey was *Setosphaeria rostrata*, which was found associated with leaf blight symptoms in wheat. This result was supported by a survey that found *S. rostrata* causing blights, spots and blotches in wheat leaves in different growing area in India (Singh et al., 2001). In this survey *F. equiseti* was the only *Fusarium* species recovered from the ear samples. In Europe, this species is one of the *Fusarium* species that cause Fusarium head blight (FHB) and it is known to produce diacetoxyscirpenol (DAS) and zearalenone (ZEA) toxins (Slivia & Ruth 2010; Thrane 2001; Bottalico & Perrone 2002). Other *Fusarium* species that have been recovered from this study were *Fusarium nygamai*,

Fusarium oxysporum f. sp., *Fusarium necatrioides*, *Fusarium incarnatum* and *Fusarium acutatum*.

All of these species were recovered at low frequency from leaf and stem samples. *Fusarium* species isolated in this study were also reported by other analysts as potentially able to blight stem bases, roots, leaves, ears as well as kernels (Liggitt et al., 1997; Narkiewicz-Jodko et al., 2003). Besides, it is a very important pathogen causing head blight in different areas where wheat is grown; *Fusarium* pathogens were found to cause crown rot and root rot in wheat fields in Turkey and USA (Smiley et al., 2005; Tunali et al., 2008). Here, in the study, aside from *Fusarium* spp., various fungi were isolated which are considered frequent in soil and stem bases, implying that the soil environment is an important source of inoculum. These included *Alternaria*, *Pleospora*, *Botrytis*, *Cladosporium*, *Cochliobolus*, *Mucor*, *Curvularia*, *Phoma*, *Rhizopus* and other.

There were no significant differences in the pathogenicity tests of four different pathogens recovered from this study causing chlorosis and necrotic lesions in the leaf of two wheat cultivars Cooly (local variety) and W.Q.226 (adapted). *B. sorokiniana* caused the highest chlorotic percentage in leaf of local variety (66%) followed by *S. rostrata* but with no significant differences. However, *A. alternata* caused the highest chlorotic percentage (53%) in leaves of adapted variety followed by *B. sorokiniana* without any significant differences. The lowest chlorotic percentage in leaves of both varieties was caused by *F. equiseti* with significant differences. Most

diseases were causing very low or no necrotic lesions in the leaves. Among the four diseases evaluated, *B. sorokiniana* was found to cause the highest necrotic lesions in the leaves of both varieties (1.3% local and 1.9% adapted) however without significant differences. These results support a previous study on 81 wheat fields in the USA which reported *B. sorokiniana* as the most frequent pathogen isolated and the most virulent pathogen in the greenhouse pathogenicity tests (Strausbaugh et al., 2004).

The influence of agronomic practices on disease incidence in wheat was considered extensively during the 2014 survey. For instance, urea application seems to influence disease incidence as the result showed that fields receiving urea had higher stem base incidence compared to the fields without urea application. Also, foliar application with potassium and ammonium influenced the incidence of the loose smut disease. Moreover, fields fertilized with urea after 2 months from sowing recorded fewer incidences of leaf spot compared to the fields received urea after 1 month from sowing. The results support findings stating that urea applied to tomato plants resulted in a wilt caused by *F oxysporum* f. sp. *lycopersici* and increased disease mortality in a bare root Douglas-fir seedling (James 1996). In addition, Buramiai province had much higher incidence of Leaf spot disease among other provinces (F was significant at $p=0.005$). While, Thahira province had higher incidence of stem base diseases (F was significant at $p=0.010$). The lowest incidence of leaf spot was recorded from Sharqia, whilst Batinah had the lowest incidence of stem base disease.

On the other hand, although there was less data collected regarding the agronomic practices between 2009 and 2013, the available data were added to data collected in 2014 and screened with multiple regression models. Sowing method has been found to influence leaf spot incidence with the field that use mechanical sowing had more leaf spot (6.9%) than the field using manual method (3.0%). These results are supported by a study, which showed that sowing method and crop rotation have been found to affect the occurrence of *Fusarium* on wheat (Tonev et al., 2008). In addition, province was also found to influence the incidence of leaf spot with Sharigia having the highest disease incidence (6.7%) and Batinah having the lowest disease incidence (1.2%). Variety also influenced leaf spot with W.Q.302 being the most susceptible in the field (15.9%); whilst W.Q.101 was the lowest susceptible variety, with 1.2% leaf spot disease (1.2%). Interestingly all local varieties screened in this study had lowest disease incidence compared to adapted variety.

Stem base diseases were influenced by years, sowing method and irrigation during the same period and same growth stage. Fields using drip irrigation system had highest stem base incidence with 1.3%, whilst fields irrigated with either flood or sprinkler irrigation had almost the same incidence with 0.33% and 0.32% respectively. Crops under irrigated condition become denser. This can modify the surrounded microclimate, promoting diseases development and pathogen sporulation. Work carried out in Canada showed that foliar diseases in wheat increased in the presence of sprinkler irrigation (Turkington et al., 2004). Fields that use mechanical sowing had highest

stem base disease comparing to manual sowing, with 1.6% and 0.31%, respectively.

The distribution and varying degree of severity justifies that there is a slow but progressive increase in the disease scenario and this situation may change under certain environmental conditions. Result obtained from this survey will help to identify the main diseases threatening wheat production in Oman. In addition, survey findings can help in determining the priority in disease problems, plan for future research to assess the economic importance and to contrast environment models for yield loss caused by disease as well as developing effective integrated disease management strategies. The results from this study demonstrate for the first time the influence of the agronomic factors on stem, leaf and ear disease occurrence in Omani wheat and pathogens associated with them.

Chapter 3

3. ECONOMIC LOSSES DUE TO EYESPOT DISEASE AND MODELLING OF PROFITABILITY AND UNCERTAINTY

3.1. INTRODUCTION

Eyespot is considered to be the most damaging fungal stem base disease of winter wheat, barley and rye in temperate regions (Crous et al., 2003; Hardwick et al., 2001; Cook et al., 1991). Wheat is economically the most important arable crop occupying approximately 41% of the arable land area in 2015 (DEFRA, 2015). There are two common pathogens that cause eyespot in the UK, *Oculimacula yallundae* (known as “W” type) and *O. acuformis* (known as “R” type; Crous et al., 2003). Scott et al. (1975) suggested that W-type (*O. yallundae*) isolates are more pathogenic to wheat whilst the R-type (*O. acuformis*) isolates are equally pathogenic to all cereal species. Eyespot disease symptoms appear as eye-shaped lesions with a diffused brown margin at the end of the season. Wheat grain size and final yield are reduced due to the pathogens blocking the vascular tissues and impeding water and nutrient movement in the plant (Ray et al., 2006).

Agronomic and environmental factors play important roles in the severity of eyespot disease. Rainfall is responsible for spreading the conidia from the soil debris to the susceptible coleoptile of the host plants. Once the infection plaques are formed by the fungal pathogen on the plant host, mycelium penetrates successive leaf sheaths reaching the stem exhibiting by this time typical browning and lesions early in the season. Typically, initial inoculum and favourable conditions in March and April lead to a peak of sporulation that declines as temperature increases, with little sporulation in June and July (Fitt et al., 1988).

The UK's mild, wet weather in the winter and cool damp weather in the spring favour the disease and commonly crop rotation and late sowing are employed as cultural control methods to reduce disease severity in high-risk regions (Cook, 1993). An accumulated risk score assessment to predict the development of eyespot has been developed by AHDB-HGCA. The assessment allows farmers to use cultivation method, sowing date, previous crop, soil type and eyespot incidence at stem extension as factors to assess risk of eyespot epidemics (Burnett and Hughes, 2004). Introduction of new wheat varieties and fungicides to treat for eyespot were later used to improve the risk assessment and assess the economic loss due to eyespot disease (Burnett et al., 2012).

3.1.1 LOSSES ASSOCIATED WITH EYESPOT DISEASE

Yield loss due to eyespot has been difficult to quantify due to the lack of correlation between early disease severity (GS31/32) and disease severity or yield at harvest (Scott & Hollins, 1978; Goulds & Fitt, 1991). Furthermore, early work on yield loss caused by eyespot between 1970 and 1990 is unclear on which fungal species was causing the disease since *Oculimacula* spp. at this time were not shown as taxonomically distinct. However, three years trials with a Consort variety showed a significant negative correlation between the yield loss and % incidence plant affected of *Oculimacula aciformis* at GS69 (Ray et al., 2004).

A more recent study using single tiller measurements showed that in fact losses caused by individual pathogens were similar, 6% and 11% reduction when the disease was caused by *O. yallundae* and *O. aciformis*, respectively (Ray et al.,

2006). A study carried out in the UK investigating the importance and control of common eyespot in wheat demonstrated that there was a significant association between eyespot disease incidence and yield (Burnett & Oxley, 1996). The same study showed that yield loss was also associated with lodging although the correlation was not as strong as eyespot disease and yield, whilst a significant correlation between eyespot and lodging was shown.

3.1.2 RISK ASSESSMENT AND DECISION MAKING

Risk assessment of eyespot disease has focussed on cost effectiveness of disease treatment through early determination of an eyespot threshold level. Between the 1970s and 1980s, several disease forecasting and risk assessment schemes were developed to predict the occurrence of severe eyespot at a time when spray decisions need to be made, based on cropping history, cultivars and environmental factors (Fitt et al., 1988). To predict where it would be cost-effective to apply an eyespot fungicide treatment, a visual threshold of 20% affected shoots at stem extension has been used. Use of fungicides in response to eyespot in 58 wheat fields in UK during 1980s was investigated (Jones, 1994). In this study the threshold of disease incidence was set at 20% and crops were classified according to their response to prochloraz treatment at GS30/31. Therefore, the decision to treat the crop with fungicides was made if more than 20% of tillers were affected with eyespot. Moreover, a study to investigate the accuracy of the assessment using the threshold method concluded that although this method could identify correctly those stems that passed the threshold level, other diseased stems that did not pass the threshold would be missing and thus

the disease would still progress (Hughes et al., 1999). In addition, with the change of the cereal varieties in the UK over the last 25 years and the dominance of *O. aciformis* eyespot species over *O. yallundae*, the use of threshold scheme is debatable (Albertini et al., 2003).

Later research by Burnett and Hughes (2004) has replaced this previous approach by developing an accumulated risk score based on a number of risk factors. This approach allows farmers to use factors such as previous crop, tillage method, sowing date, soil type, expected spring rainfall and level of eyespot at stem extension to determine the risk of economically damaging eyespot and weather and judge if it is beneficial to treat or not. This risk assessment was developed to assess the need of chemical treatment in the spring. However, with the introduction of eyespot resistant varieties, and introduction of alternative fungicides to cyprodinil, a model to judge the risk of eyespot prior to drilling was required. This risk assessment was updated by Burnett et al. (2012), by predicting eyespot risk and together with a revenue calculator to account for the cost of the treatment, grain price, efficacy of the treatment and the yield loss. This two-phase approach to assess the risk of eyespot gives the grower options to select prior to drilling, based on the eyespot score: either to use an alternative field or to drill a variety of wheat with eyespot resistance. In addition, it allows the grower to decide whether to apply fungicide treatment in the spring or not, using pre-disease score in autumn and information on eyespot incidence at stem extension.

From an economic perspective, risk is a measure of uncertainty – the uncertainty of outcomes associated with decision-making. Clearly in the case of crop diseases, we will be interested in uncertainty of outcomes associated with different decisions relating to the management of disease. Risk is always a continuing concern to the risk averse farmer; farmers with greater levels of risk aversion will wish to manage uncertainty to a greater extent than those who are less risk averse (Hardaker et al., 2004). Generally, risk is less if the farmer has an idea about final outcomes (for example, a known yield distribution) but greater if the outcome is unknown (Antle, 1983). The lower the variability, the lower the risk. However, for farmers who are risk neutral, or who are risk takers there will be a tendency to push for a more profitable (on average), but more variable outcome. Farmers who are risk averse in their decision will choose options (for example, disease management programmes) that lead to less variability, even if this choice brings lower average profitability (Moschini & Hennessy, 2001).

Generally, farmers are assumed to be risk averse; however, this is not always the case. Wossen et al. (2015) reported that 65.8% of smallholder households in Ethiopia were risk averse, with the implication that the remainder were not. Assuming risk aversion, a metric such as the standard deviation is useful to show the level of variability and thus risk relating to different choices. Risk in agriculture is usually ‘high’ as the result of a decision is unclear when the decision is made and the expected outcome value often has a high level of uncertainty. As well as output variability, farmers face variations in input costs (for example, through changing oil prices), although generally variability of

profit is influenced most by final yield and output price variability. Other risks include those associated with adopting new technology, changes in agricultural policy, exchange rate fluctuations, among others (Hardaker, 2006). Crop disease, through its effect on yield is clearly suited to risk analysis, as there are different methods of management that will have variable outcomes depending on a wide range of factors.

3.1.3 GROSS MARGIN IMPORTANCE

Disease is only one variable that farmers have to take account of in their decision making. One effective way of bringing together these other factors is to use what is termed the ‘gross margin’ – the output value (price times yield) less the direct variable costs of production: seed, fertiliser and crop protection including disease management costs. Where the labour and machinery vary with the decision being made, for example, where an agricultural contractor is used to complete spraying operations, these contract costs can be included as part of the gross margin (Nix, 2010).

Gross margin is a widely used technique by UK farm managers to plan and analyse their farm businesses, at least since the 1970s (Cassey, 1973). In addition, the universal language of the gross margin can reduce communication problems among researchers and farm managers (Nix, 1979). Furthermore, farmers can easily understand and calculate gross margins and the processes involved. Gross margins can guide the farm manager to select the right enterprises (the mix of different crops, livestock and other enterprises on the farm) and compare different technologies. The majority of the research that

addresses eyespot disease has mainly considered yield loss, with less attention paid to costs and wider output consequences of different treatments. Here we use descriptive statistics such as ‘mean’ and ‘standard deviation’ to analyse decisions of whether to treat or not to treat for a range eyespot disease trials run between 2004 and 2014 in different parts of the UK.

3.2 AIM AND OBJECTIVES:

The overall aim of this study is to improve economic decision-making relating to different eyespot management strategies.

Objectives

- 1) To assess if the treatment cost of eyespot control is recovered through yield response of the crop.
- 2) To quantify the effect of other variables on the final yield and the gross margin.

3.3 METHODOLOGY

3.3.1 DATA COLLECTION

This study used historical data collected through previous research projects on fungicide efficacy against eyespot disease by the University of Nottingham, Harper Adams University, as well as The Arable Group research (TAG). Site details including trial code, regions, soil type, previous crop, tillage, and sowing date were recorded in the database as shown in Table 7.1 in the Appendix.

Experiments were positioned across various locations between 2004 and 2014. Experimental field locations and their GPS coordinations are shown in Table 7.2 in the Appendix. Data of natural infection or artificially inoculated eyespot efficacy trials used in this study is shown in Table 7.3 in the Appendix. Moreover, fungicide treatments that have been tested during the period of 2004 to 2014 as well as field rate per hectare of each fungicide were recorded in the database that are presented in Table 7.4 of the Appendix.

From the database, we can quantify the variables needed to calculate the gross margins. The available data to perform gross margin calculation were yield and rates of fungicides application; however, other variable costs were provided from Agro Business Consultants Ltd. (ABC) as of November 2014.

3.3.2 FINANCIAL ANALYSIS DATA

Chemical cost

BASF and Agrii supplied the 2015 average price of the chemicals assessed in all trials. All chemical product names, along with their active ingredients and costs, are presented in Table 3.1.

Table 3-1: Fungicides trade names, active ingredients and price per litre for 2015.

Chemical Names	Active Ingredients	Price per litre £/l
Adexar	Epoxiconazole and Fluxapyroxad	35
Amistar	Azoxystrobin	34
Aviator 235xpro	Bixafen and Prothioconazole	42
Bravo	Chlorothalonil	6.5

Capalo	Epoxiconazole, Fenpropimorph and Metrafenone	31
Cerix	Epoxiconazole, Fluxapyroxad and Pyraclostrobin	27
Chord	Boscalid and Epoxiconazole	26
Ennobe	Epoxiconazole and Prochloraz	18
Enterprise	Boscalid and Epoxiconazole	26
Flexity	Metrafenone	65
Ignite	Epoxiconazole	20
Intrex	Fluxapyroxad	25
Keystone	Epoxiconazole	40
Librax	Fuxapyroxad and Metconazole	36
Nebula	Boscalid, Epoxiconazole and Pyraclostrobin	26
Opus	Epoxiconazole	20
Proline	Prothioconazole	50
Seguris	Isopyrazam and Epoxiconazole	42
Tracker	Epoxiconazole and Boscalid	26
Unix	Cyprodinil	14
Vertisan	Penthiopyrad	28
Xemium	Fluxapyroxad	24

Application cost

Chemicals were used in the trials at different rates of application. To calculate the cost of fungicide per hectare, the exact rates of application were multiplied by the actual cost of the fungicide as shown in Table 3.2.

Table 3-2: Fungicides, their rate of application and cost per hectare.

Fungicides	Field (l/ha ⁻¹)	rate	Price (£/ha ⁻¹)
Adexar	1		35
Adexar	0.67		23
Adexar	0.75		26
Adexar	1.33		47
Adexar	2		70
Adexar + Bravo	1+1		42
Aviator 235 xpro + Bravo	1+1		49
Aviator 235 Xpro	1		42
Aviator 235 Xpro	0.42		18
Aviator 235 Xpro	0.84		35
Aviator 235 Xpro	1.25		53
Capalo	1		31
Cerix	1		27
Cerix	1.5		41
Chord	1		26
Ennobe	1		18
Enterprise	0.83		22
Enterprise	1		26
Enterprise	1.675		44
Enterprise	2.5		65
Ignite	0.75		15
Ignite	1		20
Imtrex	1		25
Librax + Bravo	1+1		43
Nebula	0.83		22
Nebula	1		27
Nebula	1.675		45
Nebula	2.5		67
Opus	0.5		10
Opus	0.67		13
Opus	0.75		15
Opus	1		20
Opus + Unix	0.5+0.67		19

Opus + Flexity	0.5+0.25	26
Opus + Flexity	0.5+0.5	43
Opus + Amistar	0.75+0.5	32
Proline	0.4	20
Proline	0.54	27
Proline	0.6	30
Proline	0.8	40
Proline	1	50
Seguris	0.33	14
Seguris	0.66	28
Seguris	1	42
Seguris +Bravo	0.8+1	40
Tracker	0.5	13
Tracker	0.75	20
Tracker	1	26
Tracker	1.5	39
Tracker + Bravo	1+1	33
Tracker + Bravo	1.5+1	46
Tracker + Adexar	1+1	61
Tracker + Xemium	0.5+0.5	25
Unix	0.5	7
Unix + Opus	0.5+0.5	17
Unix + Opus	1+0.5	24
Vertisan + Ignite + Bravo	1+0.75+1	50
Xemium	0.67	17
Xemium	1	25
Xemium	1.33	33
Xemium	1.5	37
Xemium	2	49

Other variable costs

Other variable costs were as farm standard practice and assumed to be the same across all trials. Data were obtained from Agro Business Consultants Ltd (ABC) as of November 2014 (Table 3.3).

Table 3-3: Other variables and its prices (ABC, 2014)

Other Variable costs	Price (£/ha ⁻¹)
Seed	53
Nitrogen	165
Application charge (contractor)	27

Grain prices

The price of wheat has been derived from Agro Business Consultants Ltd as £139/tonne. This is an estimated average ex-farm selling price from the 2015 harvest.

3.3.3 DATA ANALYSIS

Descriptive statistics such as ‘mean’ and ‘standard deviation’ were used to gain an overview of the data. From an economic perspective, the mean is the expected outcome and the standard deviation is the variation around this outcome, our measure of risk. Assuming normal distribution +/-2 standard deviations represents 95% of a distribution.

3.4 RESULTS

3.4.1 YIELD MEANS OF INOCULATED AND NATURAL INFECTED TRIALS 2004-2014

The yield means for both treated and untreated eyespot disease inoculated trials are shown in Figure 3.1. Average yield for the treated trials was 9.2 t/ha⁻¹; for the untreated trials was 8.4 t/ha⁻¹. Yield means for the treated and untreated

trials were highest in 2004, at 10.8 t/ha⁻¹ and 10.2 t/ha⁻¹ respectively; lowest mean yield was 8.5 t/ha⁻¹ in 2006 for treated trials and 7.2 t/ha⁻¹ in 2012 for untreated. The highest yield variation was found in treated trials for 2006 with and untreated in 2007 showing ± 2 SDs of 2.1 t/ha⁻¹ and 2.6 t/ha⁻¹ respectively. All other years of treated trials had ± 2 SDs between 0.7 and 1.4 t/ha⁻¹, while other years in the untreated trials had ± 2 SDs between 0.9 and 1.8 t/ha⁻¹. Student *t* tests showed that the yield of all fungicide treated trials was significantly different as compared to untreated trials of the same year except the yield year of 2005. The average yield of significant fungicide treated year was 9.2 t/ha⁻¹; whilst the average yield of significant untreated year was 8.4 t/ha⁻¹. Variability was generally lower within individual years in the naturally infected trials; however, the high mean yield recorded in 2009 had a relatively large variability (Figure 3.2). The average yield of treated naturally infection trials was 10.1 t/ha⁻¹, however the average of untreated trials was 9.7 t/ha⁻¹.

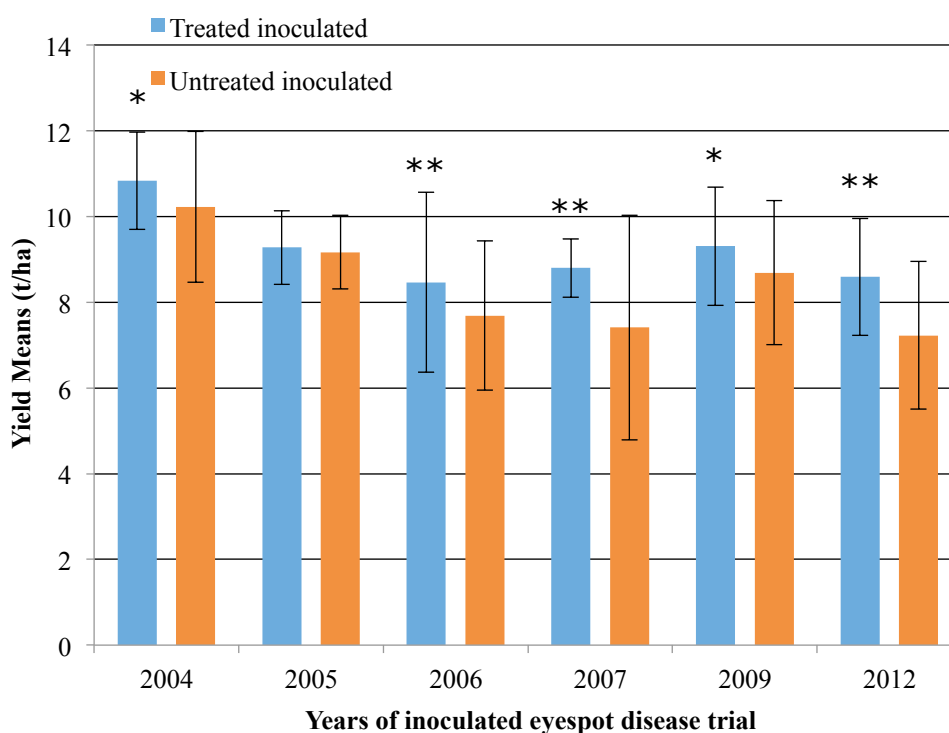


Figure 3-1: Yield means for treated and untreated eyespot disease inoculated trials between 2004 and 2012 (± 2 SDs). Yield means of treated inoculated trials of 2004, 2006, 2007 and 2012 were significant comparing to untreated inoculated trials in the same years. The highest yield was recorded in 2004 in both treated and untreated trials.

Untreated ($n-I$), 2004=15, 2005= 15, 2006= 15, 2007=11, 2009= 11 and 2012=11. Treated ($n-I$), 2004= 111, 2005= 143, 2006= 127, 2007=67, 2009=29 and 2012=125.

*Signifiant codes : 0 '****' 0.001 '**' 0.01 '*' 0.05.

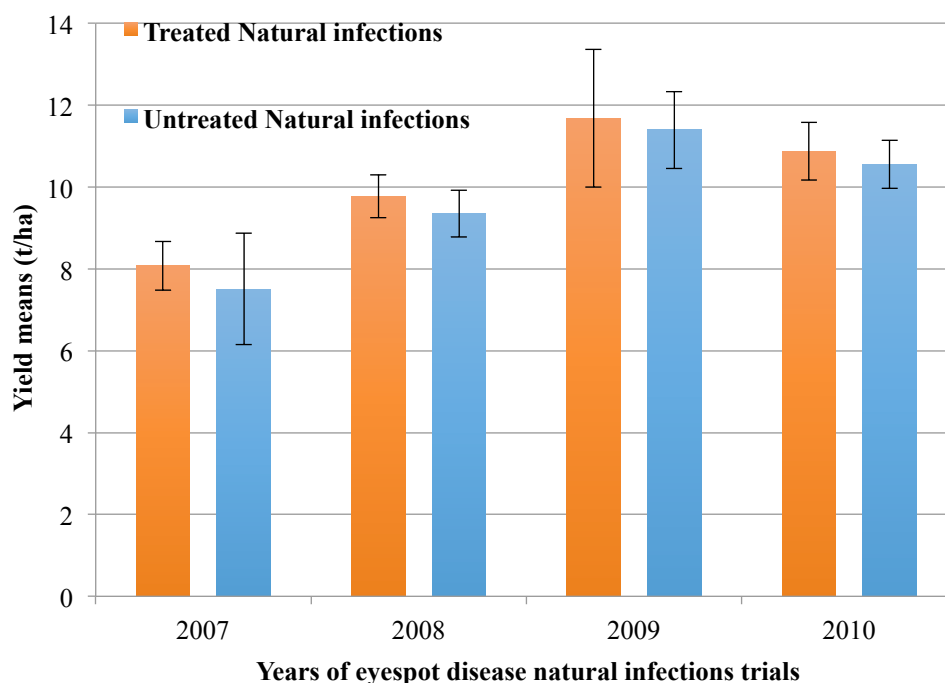


Figure 3-2: Yield means for eyespot disease naturally infected trials between 2007 and 2010 (± 2 SDs). Yield means of all treated natural infection trials were higher comparing to untreated natural infection trials in the same years. The highest yield was recorded in 2009 in both treated and untreated trials.

Treated ($n-1$), 2007= 67, 2008= 13, 2009= 31, and 2010= 31. Untreated ($n-1$), 2007= 11, 2008= 2, 2009= 7, and 2010= 7.

3.4.2 GROSS MARGIN MEANS OF INOCULATED AND NATURALLY INFECTED TRIALS 2004 - 2014

The gross margin means of inoculated trials for both treated and untreated years are shown in Figure 3.3. Across all years, the gross margin mean of the treated trials was £1021.9 ha⁻¹; for the untreated trials it was £950.1 ha⁻¹. The treated and untreated trials in 2004 had the highest gross margin on average, at

£1254.1 ha⁻¹ and £1203.5 ha⁻¹ respectively. The lowest mean gross margin was recorded in 2012 (£913.3 ha⁻¹) for treated trials and also in 2012 (£786.8 ha⁻¹) for untreated. Thus, variability is greater between years than within the trials. The value of ± 2 SDs was used to demonstrate the variation in the results. The highest variation was in 2008 (treated) with ± 2 SDs of £291.6 ha⁻¹ and 2007 in untreated trials with ± 2 SDs of £364.4 ha⁻¹. All other years of treated trials had ± 2 SDs between £93 ha⁻¹ and £180.2 ha⁻¹, while the other years in the untreated trials had ± 2 SDs ranged between £119 ha⁻¹ and £243.3 ha⁻¹. There were significant differences in 2005, 2007 and 2012 between treated and untreated trials; the average of the significant trials was £973 ha⁻¹ whilst the average in the untreated trials was £885 ha⁻¹. The gross margin mean of the treated naturally infected trials was £1135.2 ha⁻¹, whilst the gross margin mean for the untreated trials was £1130.4 ha⁻¹, as shown in Figure 3.4. The highest value was recorded in untreated trials in 2009 at £1366.1 ha⁻¹. The value for ± 2 SDs varied in treated natural infection trials from £74 ha⁻¹ in 2008 to £233 ha⁻¹ in 2009. However, the ± 2 SDs values in untreated natural infection trials vary from £79.6 ha⁻¹ in 2008 to £189.5 in 2007 ha⁻¹. There were no significant differences between treated and untreated trials in natural infection.

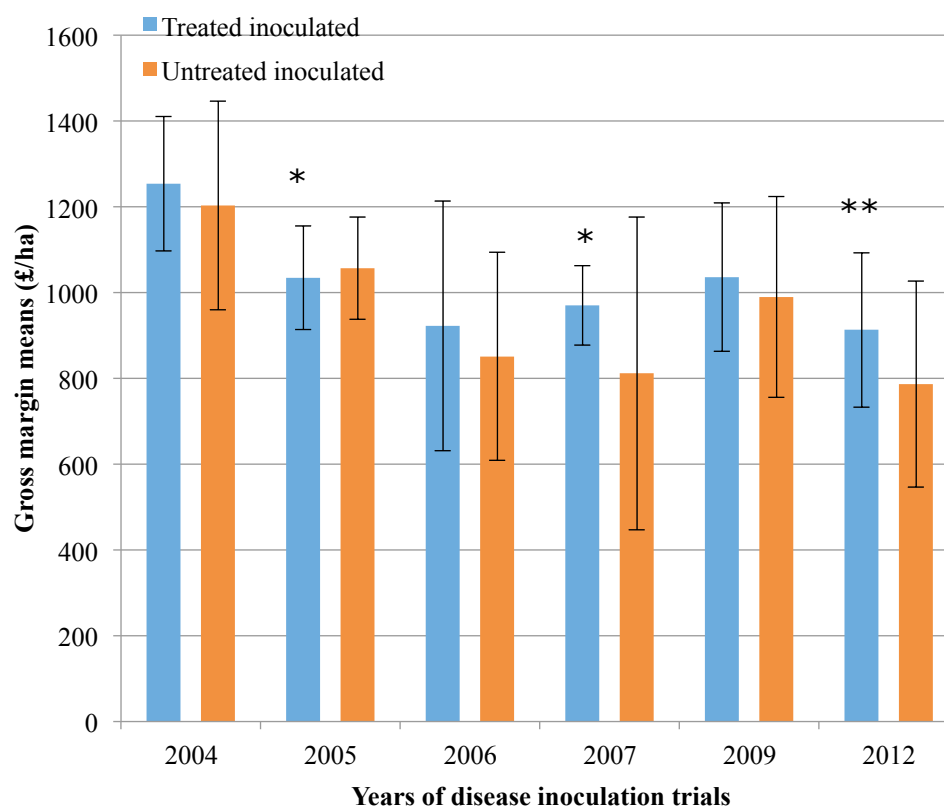


Figure 3-3: Gross margin means for treated and untreated inoculated trials between 2004 and 2012 (± 2 SDs). Gross margin means of treated inoculated trials of 2005, 2007 and 2012 were significant comparing to untreated inoculated trials in the same years. The highest gross margin was recorded in 2004 in both treated and untreated trials.

Untreated ($n-1$), 2004=15, 2005= 15, 2006= 15, 2007=11, 2009= 11, and 2012=11. Treated ($n-1$), 2004= 111, 2005= 143, 2006= 127, 2007=67, 2009=29 and 2012=125. *Significant codes: 0 '****' 0.001 '**' 0.01 '*' 0.05.

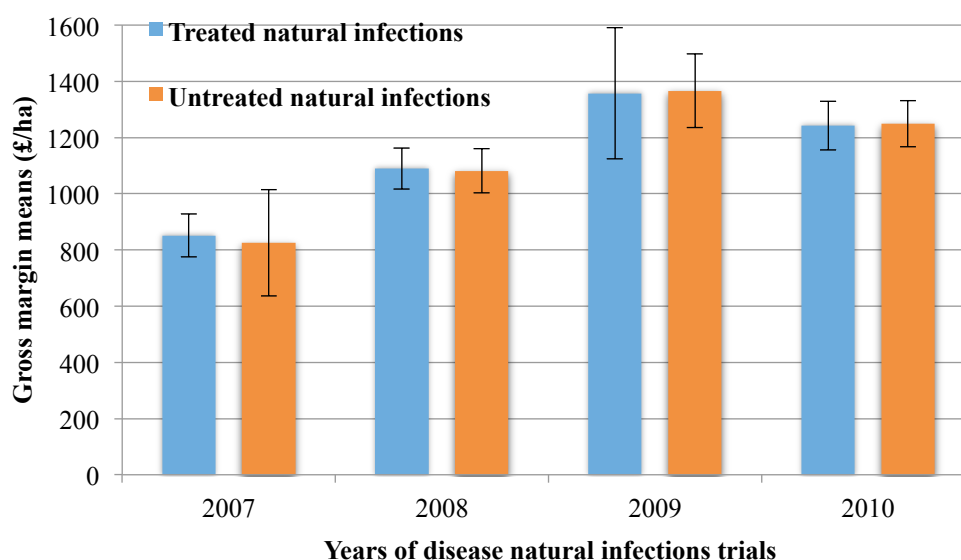


Figure 3-4: Gross margin means for treated and untreated naturally infected trials between 2007 and 2010 (± 2 SDs). The gross margin of both treated and untreated natural infection trials is not significant. The highest gross margin was recorded in 2009 in both treated and untreated trials.

Treated ($n-1$), 2007= 67, 2008= 13, 2009= 31, and 2010= 31. Untreated ($n-1$), 2007= 11, 2008= 2, 2009= 7, and 2010= 7.

3.4.3 THE EFFECT OF VARIOUS FUNGICIDE TREATMENTS ON EYESPOT DISEASE INDEX AT GS70/80 IN INOCULATED AND NATURALLY INFECTED TRIALS

The variation effects of lower and higher doses of three different fungicides upon disease index (DI) at GS70/80 in the inoculated trials were investigated as shown in Figure 3.5. The DI of each treatment and control condition of each trial year was calculated as explained in the methodology section in chapter 4. It was found that Tracker application resulted in a clear dose response, with

disease index decreasing as the dose increased from half to full rate and both doses reduced DI compared to the control (no fungicide treatment at GS30/31). However, Opus and Proline 275 were not shown to produce a dose response.

The highest variation was found in Opus at 1 l ha⁻¹ with ± 2 SDs of DI 43.5% and the lowest ± 2 SDs in Tracker 1.5 l ha⁻¹ with DI 34.3%. Tracker at 1.5 l ha⁻¹ plus Opus at 0.5 l ha⁻¹ significantly reduced the disease index compared to the control. A similar pattern was seen with the effect of different fungicide treatments on the disease index at natural infection trials. All three fungicides reduced DI in the treated trials comparing to the untreated ones as shown in Figure 3.6. Proline at 0.6 l ha⁻¹ and Opus at 0.5 l ha⁻¹ significantly reduced the DI compared to the untreated control.

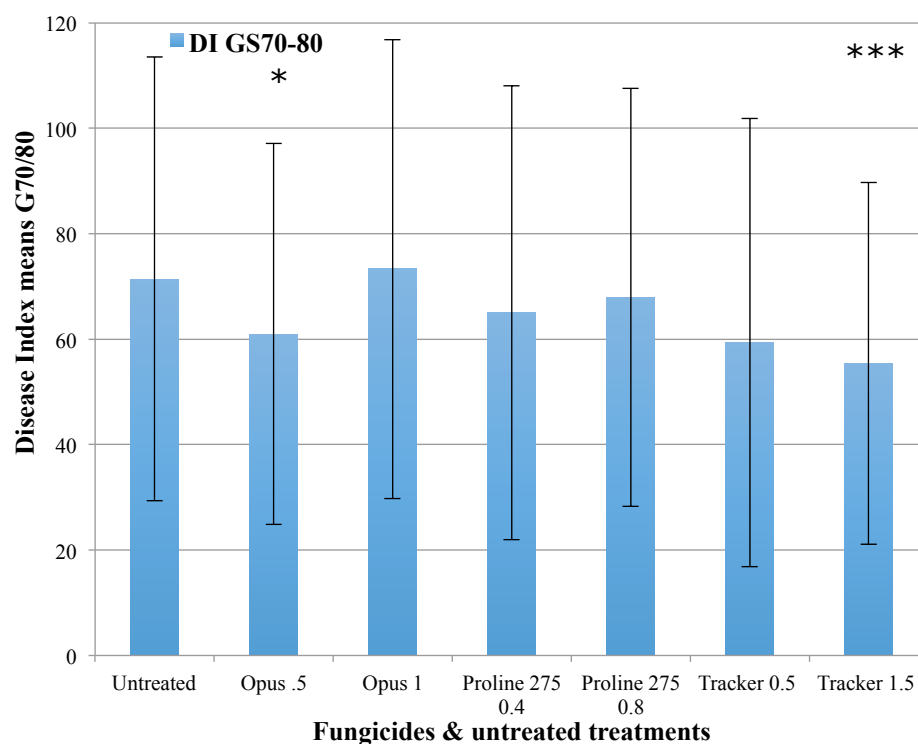


Figure 3-5: Disease index at GS70/80 means of different fungicides used at GS 31 in inoculated trials between 2004 and 2014 (± 2 SDs). Tracker at higher dose 1.5 l ha^{-1} was highly significant to decrease DI at GS70/80 compared to control. Opus at 0.5 l ha^{-1} was significant to decrease DI compared to control. Apart from Opus at 1 l ha^{-1} dose, all other fungicides decreased the disease index at GS70/80 insignificantly compared to control.

***(n-1): Opus 0.5=26, Opus 1=19, Proline 275 0.4= 31, Proline 275 0.8 =55, Tracker 0.5 = 14, Tracker 1.5= 62, Untreated=111.**

Significant codes: 0 '*' 0.001 '***' 0.01 '**' 0.05.**

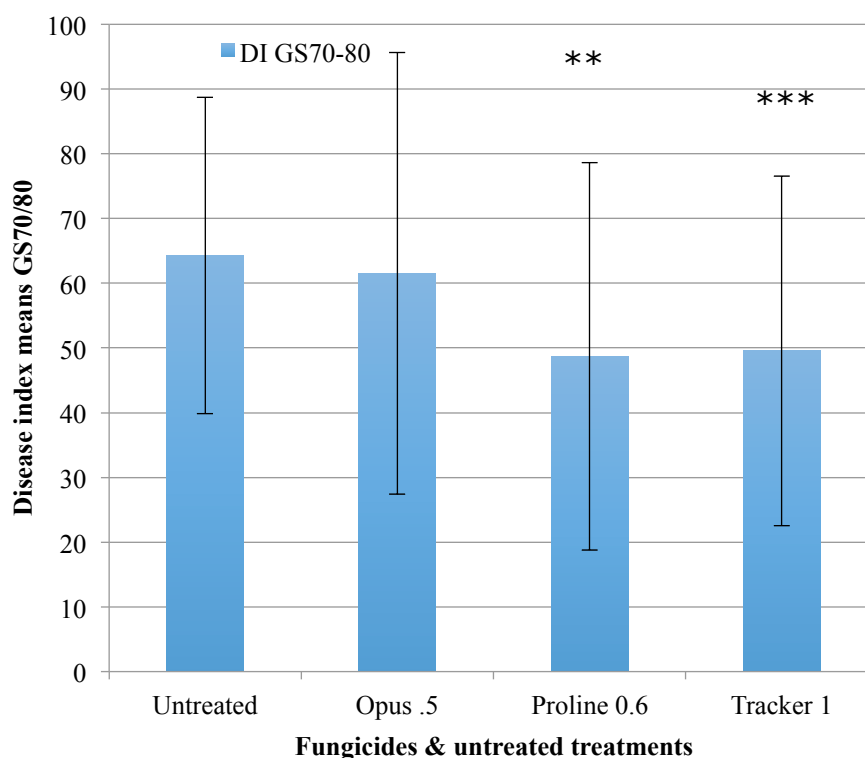


Figure 3-6: Disease index means at GS70/80 of different fungicides used at GS 31 in natural disease infection trials between 2007 and 2010 (± 2 SDs). Tracker at 1 l ha^{-1} and Proline at 0.6 l ha^{-1} was significant to decrease DI compared to untreated. Opus at 0.5 l ha^{-1} reduced DI at GS70/80 insignificant compared to control.

***(n-1): Opus 0.5=15, Proline 275 0.6=26, Tracker 1= 34, and Untreated = 30.**

Significant codes: 0 '*' 0.001 '**' 0.01 '*' 0.05.**

3.4.4 THE EFFECT OF VARIOUS FUNGICIDE TREATMENTS ON YIELD IN INOCULATED AND NATURALLY INFECTED TRIALS

The effect of various fungicide treatments on yield of wheat in inoculated trials is shown in Figure 3.7. All fungicide treatments increased yield apart from Opus at 1 l ha^{-1} and Tracker at 0.5 l ha^{-1} compared to the untreated control. The

average yield of significant treatments was 9.5 t/ha whilst the average of the untreated was 8.4 t/ha. The yield in the naturally infected trials was also increased due to fungicide treatment as shown in Figure 3.8. However, Opus at 0.5 l ha⁻¹ was the only fungicide that significantly increased yield having an average of 11 t/ha compared to 9.5-t/ha average yield of the untreated control.

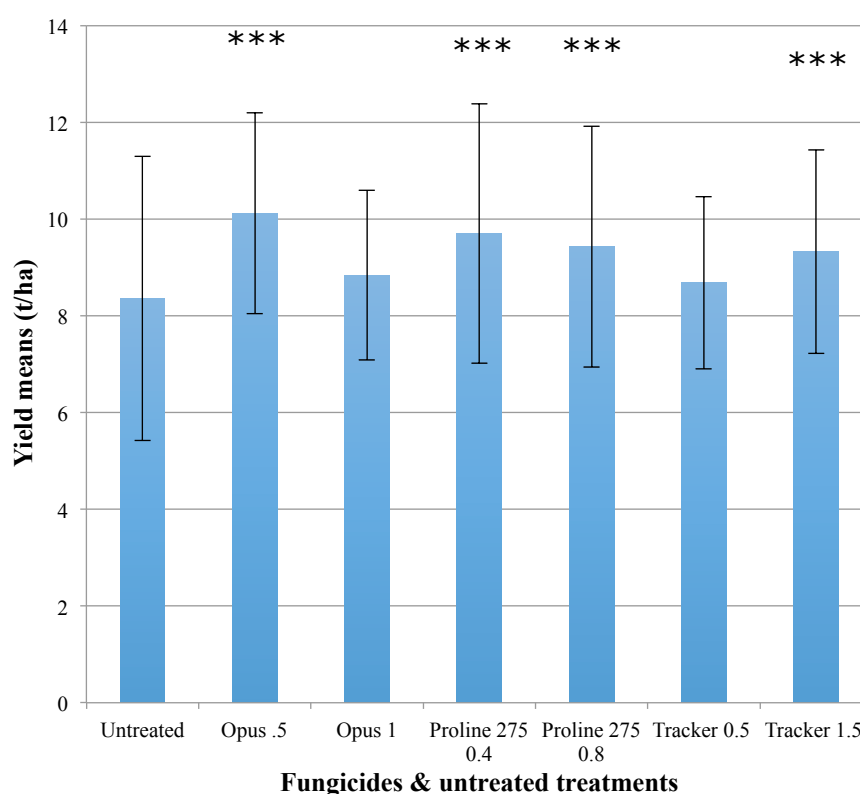


Figure 3-7: Yield means of different fungicide treatments in inoculated trials between 2004 and 2014 (± 2 SDs). Tracker at higher dose 1.5 l ha⁻¹, Proline 275 at 0.5 and 0.8 l ha⁻¹ and Opus at 0.5 l ha⁻¹ dose increased yield significantly compared to control. Opus 0.5 and Tracker 0.5 increased yield compared to control but not significantly.

***(n-1): Opus 0.5=26, Opus 1=19, Proline 275 0.4= 31, Proline 275 0.8 =55, Tracker 0.5 = 14, Tracker 1.5= 62, Untreated=111.**

Significant codes: 0 '' 0.001 '**' 0.01 '*' 0.05.**

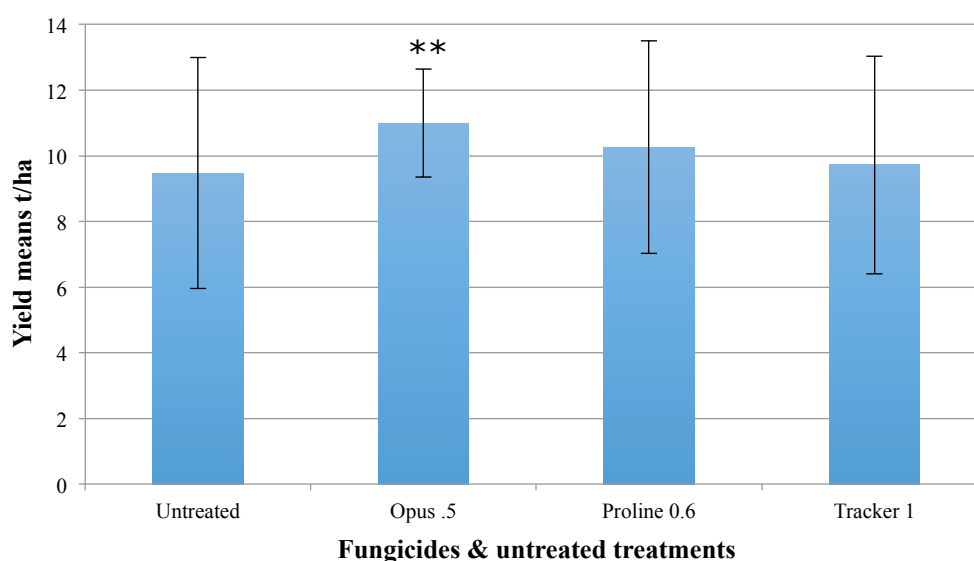


Figure 3-8: Yield means of different fungicide treatments in natural infection trials between 2007 and 2010 (± 2 SDs). Opus at dose of 0.5 l ha^{-1} was the only fungicides to increase yield significantly compared to control. Other fungicides increased yield in comparison to control but not significantly.

***(n-1): Opus 0.5=15, Proline 275 0.6=26, Tracker 1= 34 and Untreated = 30.**

Significant codes: 0 '*' 0.001 '***' 0.01 '**' 0.05.**

3.4.5 THE EFFECT OF VARIOUS FUNGICIDE TREATMENTS ON GROSS MARGIN IN INOCULATED AND NATURALLY INFECTED TRIALS

The gross margin from each of the fungicide treatments in both inoculated and natural infection of eyespot trials were investigated to check if the fungicides were worth their costs. The cost of the fungicides and the cost of the application were also considered within the gross margin. The variations among the treatments were represented by standard deviations, as before. The

average price of £1030.8 ha⁻¹ treatments was found to be worth the cost as shown in Figure 3.9. Opus at 0.5 l ha⁻¹ and Proline 275 at 0.4 l ha⁻¹ had the most favourable gross margins. The lowest variation was found with Opus at 1 l ha⁻¹ treatment with risk associated with getting a gross margin of £244.4 ha⁻¹ whilst the highest variation was found with the untreated control with risk associated with getting a gross margin of £407.9 ha⁻¹. Moreover, all fungicides except Opus at 1 l ha⁻¹ and Tracker at 0.5 l ha⁻¹ returned significantly different gross margins compared to the untreated. At higher dose Tracker returned a better gross margin than at the lower dose; however, the lower dose did show less variation. On the other hand, a different pattern was seen with fungicide treatments in the natural infection trials (Figure 3.10). The lowest gross margin was returned from Tracker at 1 l ha⁻¹ treatment with £1080.2 ha⁻¹ whilst the highest returned by Opus 0.5 l ha⁻¹ with £1273.3 ha⁻¹.

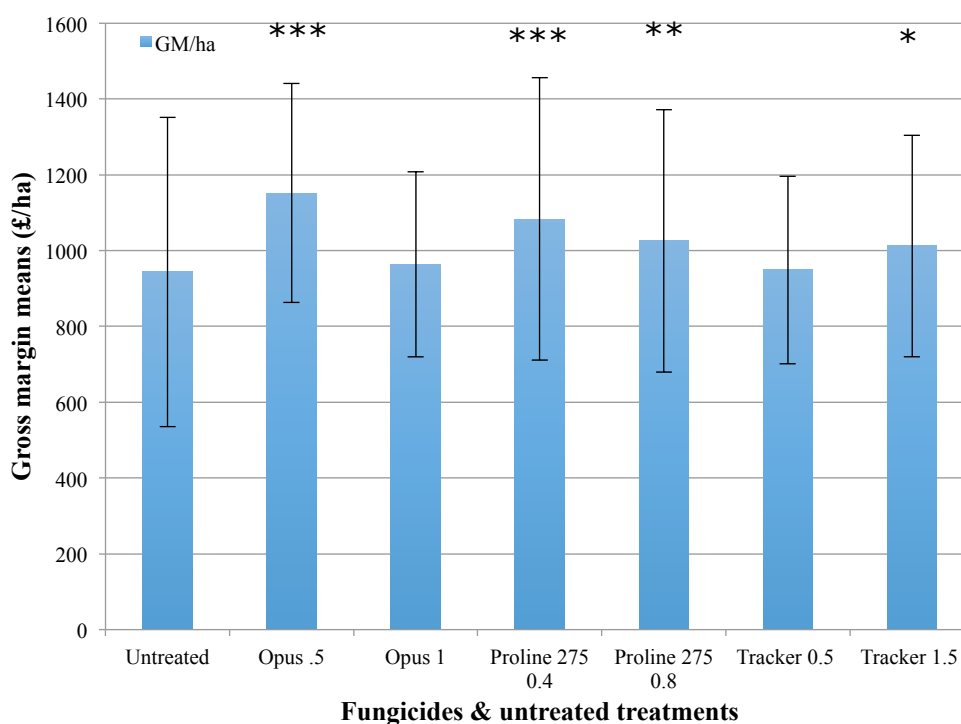


Figure 3-9: Gross margin means of different fungicide treated and untreated inoculated trials between 2004 and 2014 (± 2 SDs). Tracker at higher dose 1.5 l ha^{-1} , Proline 275 at 0.5 and 0.8 l ha^{-1} and Opus at 0.5 l ha^{-1} dose significantly increased gross margin compared to control. Opus 0.5 and Tracker 0.5 increased gross margin compared to control but not significantly.

*(n-1): Opus $0.5=26$, Opus $1=19$, Proline 275 $0.4=31$, Proline 275 $0.8=55$, Tracker $0.5=14$, Tracker $1.5=62$, Untreated= 111 .

*Significant codes: 0 '****' 0.001 '***' 0.01 '**' 0.05 .

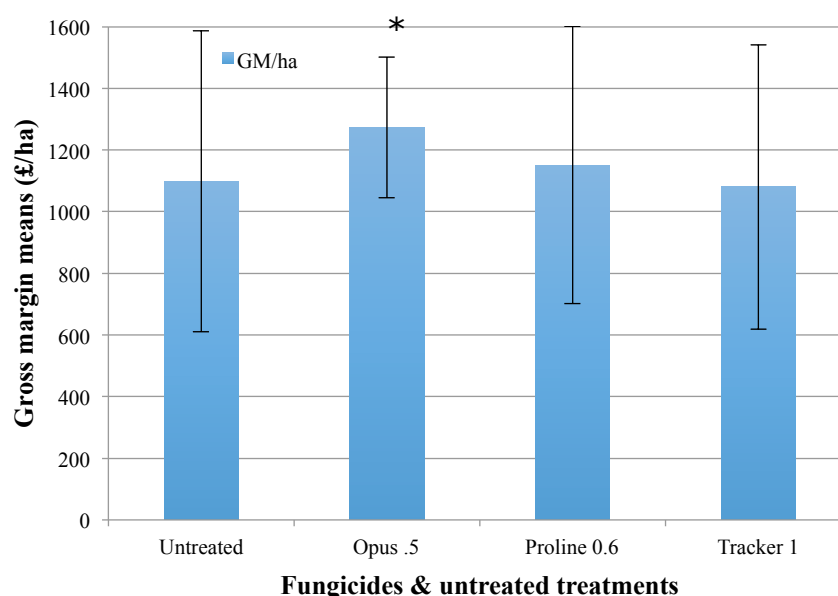


Figure 3-10: Gross margin means of different fungicide treated and untreated natural disease infection trials between 2007 and 2010 (± 2 SDs). Tracker at dose 1 l ha^{-1} returned lowest gross margin while Opus at 0.5 l ha^{-1} dose returned highest gross margin but not significantly compared to other fungicides and untreated.

***(*n-I*): Opus 0.5=15, Proline 275 0.6=26, Tracker 1= 34, and Untreated = 30.**

Significant codes: 0 '*' 0.001 '***' 0.01 '**' 0.05.**

3.5 DISCUSSION

The aim of this study was to improve decision-making by i) assessing whether treatment cost of eyespot control is recovered through yield response of the crop and ii) to assess the effect of fungicide treatment on risk. In terms of reducing eyespot disease at GS70/80, all treatments apart from Opus at 1 l ha^{-1} were shown to have an effect. This was expected as all of the fungicides assessed are known to have a degree of activity against eyespot disease. Opus (epoxiconazole) at higher dose was found to be insignificant in inoculated trials

under high disease pressure whilst under lower dose it reduced DI of eyespot significantly under low disease pressure only. A previous study by Ray et al. (2004) found Opus to reduce disease index by only 2.6% having a very small effect against eyespot. Opus at 1 l ha⁻¹ was shown to be less effective against eyespot and was classified as a part of untreated category (Burnett & Hughes, 2004). In France, a poor control has been noted with Opus against eyespot disease; particularly its activity against *O. acufomis* species was found very low (Leroux, 1998).

In this study, Tracker was found to have a clear dose response; with disease index decreasing as the dose increased. This result indicates that at a higher dose Tracker, which is a mixture of epoxiconazole and boscalid, achieved greater yield and returned better gross margin. Proline (prothioconazole) was found to be highly successful in reducing disease index and increasing yield previously (Burnett, 2005). Also, Burnett and Hughes (2004) found Proline 275 to be more effective than Opus and Unix mixtures. The results in this study indicate the effectiveness of Proline 275 to be the same as that found in literature. Opus treatment at 0.5 l ha⁻¹ reduced DI significantly and returned better gross margin and yield compared to Proline 275 treatment at 0.4 l ha⁻¹, and no dose response was identified for either treatment. This was not expected for Opus since it has lower activity against eyespot. Also, the results on Proline 275 are unexpected and in contrast to results from Burnett (2005), although at higher doses Proline 275 were most effective in reducing disease index. The better gross margin returned by Opus comparing to Proline 275 may be due to the rate of Proline 275 at 0.4 l ha⁻¹ – a cost of £20 ha⁻¹ - double the cost of

Opus at 0.5 l ha^{-1} . In this study, we only examined the effects of increasing rate of treatment in three different fungicides used in inoculated trials. Under these conditions only Tracker showed a clear dose response used from 0.5 l ha^{-1} to 1.5 l ha^{-1} . It is likely that under natural infection other diseases like *Septoria* and rust may have also been present at higher levels and dose responses may not be the same as under inoculation where eyespot disease predominates under higher severity.

Within this study the use of fungicides against eyespot and their economic effect upon DI at GS70/80 and related responses in yield and gross margin were also considered. It was identified that in both the inoculated and naturally infected trials, all fungicide treatments increased yield with most of them showing significant increases as compared to control. The gross income was calculated by multiplying the yield with grain price of $\text{£}139 \text{ ha}^{-1}$ as in 2015. The price for variables including chemical, fertilizers and contactors were subtracted. Gross margin results were quite similar to the results for yield. In most years gross margin of treated trials dominated the gross margin from untreated trials. A study to estimate the economic benefits of alternative pesticides usage scenarios on wheat production in the UK, found that if pesticides used were reduced by two thirds, gross margin per hectare would not be effected under a grain price of $\text{£}75 \text{ ha}^{-1}$ (Webster et al., 1999). However, gross margin dropped significantly if pesticides were removed completely.

Most fungicides also significantly improved gross margins with the most favourable return from Tracker at the higher dose rate. This is in agreement

with the published literature showing that fungicide treatments play an important role in increasing yields (Cook, 1980; Ray et al., 2004). In Sweden, a single treatment against eyespot at GS31/33 between 1977 and 2005 with benzimidazoles in early years and with pyrimidines in later years improved yield by 320kg/ha on average due to few years that had severe attack with eyespot (Wilk, 2009). In addition, the mean yield response to early treatment with eyespot fungicides was higher (1050 kg/ha) when average of eyespot index greater than 35%, whilst it was only 190 kg/ ha⁻¹ when eyespot index was less than 10% (Wilk, 2009).

In this study the yield means of treated inoculated and natural infections trials dominated the yield means of untreated inoculated and natural infections trials during all of the years included in the analysis. As well as the range of standard deviation (+/-2) was lower in most years of treated trials except 2006 in inoculated trials and 2009 in natural infection trials. Although yield is an important consideration for most farmers, it is also important to consider gross margins from a management perspective. As with yield, the gross margin means for inoculated trials was always higher than the gross margin for untreated inoculated trials except for 2005. Moreover, the range of (+/- 2) standard deviation of inoculated treated trials was also lower in most years than the range of untreated trials except in years 2005 and 2006.

However, in the situation where the naturally infected data was assessed, fungicide control was not shown to be significant in affecting gross margin. Nevertheless, treatment with a fungicide on average reduced the level of

variation, so although the average gross margin for untreated is competitive, treatment would still be worthwhile for the risk averse farm manager so that they would not be exposed to a gross margin variation of £189/ha (Standard deviation). This result indicates that for those farmers with risk averse behaviour, the dominant strategy will be to manage the crop with fungicides. It is interesting that all the treatments were increasing yield in both the inoculated and the naturally infected trials, however such clear results are not seen when assessing gross margins. The reason for this is thought to be because of the effect the costs associated with treatment had.

The reduction in variable cost could be very important because variable costs are correlated more directly with output unlike the fixed costs, which, by definition, remain constant regardless of output. However, this mainly depends on the risk aversion of the farm manager, although in this study the mean benefit from the treatment was marginal. However, if the crop was not treated there is then a risk of exposure to high disease severity that could cause substantial yield loss. This is well noted in the literature: e.g. Fitt et al. (1988) stated that if farm managers decided not to treat against disease, the risk of exposure to more than a 78% disease index could be detrimental to yield. Our results showed that it is worth spending more on crop management and disease control in order to enhance and maintain the yield that will return more income. There is a little information on wheat yield and profitability responses of eyespot management program that utilize fungicides for purpose than disease control. However, a study that considered the outcome of more spending on fungicides to control soybean diseases in order to gain better gross margin concluded that soybean

seed mass was increased with fungicides application and that returned more yield which in turn caused the net return to increase (Henry et al., 2011).

In addition, the results from this study showed that the level of variation among treated trials was reduced: thus, even though the mean gross margin of the untreated trials was more competitive, treatment would be still a better choice for many risk averse farmers. The relationship between the ultimate of gross margin variation and eyespot treatment has not been considered in the past. However similar research to determine the pesticide usage for site-specific weed management, found that although it was not always worth cost, there were often considerable benefits in reducing yield variability (Ritter et al., 2008). The unexplainable results were gross margin of untreated trials dominated the treated one under high disease pressure. As demonstrated within the literature review, many factors can affect the incidence of eyespot, crop yield, and ultimately the gross margin. Fungicide treatment is one such factor, however cultural practices and weather have also been demonstrated to have an effect on eyespot severity and yield losses. In addition, that could be because disease was not severe enough or other diseases were also present influencing the quantification of efficacy against eyespot of particular actives. This is well documented in the literature and using a model like the ‘threshold percentage’ may lead to incorrect identification of diseased crops that then miss the crop which needed treatment, allowing those crops to cause serious infection at later stages (Scott & Hollins, 1978; Hughes et al., 1999; Burnett et al., 2000).

Overall, not all fungicide treatments of eyespot were worth the cost. Opus at 1 l ha⁻¹ was found to be insignificant in all analysis in terms of disease index reduction and gross margin benefit. The other fungicides were found to be worth the costs, either under high disease pressure (inoculated sites) or naturally infected sites. It was most economical to apply Tracker under higher dose having the largest disease reduction and financial gain. Whilst with Opus and Proline 275 the dose response was absent and did not have any effect. When considering the risk averse nature of the farm manager, the choice of whether to treat varied, with less risk being carried by treating crops compared to missing the treatment. For the risk averse manager fungicide treatment would be worth the cost as it would reduce the higher level of disease and consequently minimise associated yield losses. It was also found that the decision not to treat resulted in an increased gross margin variation as indicated by standard deviation compared to the variation of the average fungicides.

Chapter 4

4. EYESPOT DISEASE MODELLING FOR UK

4.1. INTRODUCTION

Three factors play an important role in disease epidemics, the susceptibility of the host, the presence of inoculum of an aggressive pathogen as well as favourable environmental conditions for infection and disease development (West et al., 2012). Thus, for the initiation of disease, the spatial and temporal dispersal of plant pathogens, affected by environmental conditions, must coincide with the susceptible stage of development of the host crop in terms of phenology (growth stages). For instance, drier summer conditions reducing the breakdown of crop debris may increase inoculum availability of facultative pathogens such *Oculimacula* spp. causing eyespot disease on cereals, whilst successful infection of the host may be reliant on extended time and favourable environmental conditions for pathogen dispersal and spread during crop emergence and seedling development (West et al., 2012).

Weather forecasting engines can be used to identify optimum environmental conditions for the survival, dispersal and spread of plant pathogens to monitor disease infection risk and apply appropriate control measures. For example, temperatures of 8-20°C and high humidity associated with cloud and light rain are most favorable condition for karnal bunt caused by *Tilletia indica* to infect the ears of wheat, rye and triticale in Europe. Sansford et al. (2008) estimated the risk of pathogen infection in Europe by applying a published karnal bunt disease model based on investigation of specific phases of the pathogen lifecycle and its relation to the principal host that is wheat. The simulation showed that weather during May and June at the time of heading period or ear

emergence was most favorable for disease infection and development across Europe. Thus, it is also possible to model the effects of the climate change on disease development as well as disease effects on the crop as it develops phonologically to avoid unreliable predications (Butterworth et al., 2010, Madgwick et al., 2011).

4.1.1 EYESPOT DISEASE – EPIDEMIOLOGY AND LOSSES IN WHEAT

In the UK, eyespot caused by two closely related fungal species, *Oculimacula yallundae* and *O. acufomis* (Robbertse et al., 1995) is considered one of the most damaging disease on cereal stem-bases (Crous et al., 2003, Cook et al., 1991).

4.1.2 EYESPOT DISEASE SYMPTOMS

The early visible symptom of eyespot is brown smudge on the leaf sheath on the stem that is sometimes confused with other stem base diseases such brown foot rot caused by *Fusarium* spp. or sharp eyespot (*Rhizoctonia cerealis*) (Goulds & Polley, 1990, Burnett & Hughes, 2004). According to Blein et al. (2009), the disease has a latent period that is around six to eight weeks; often by this time symptoms are seen in the infected plant. Later in the season, the eye-shaped elliptical lesion does appear usually below the first node and symptoms become even clearer (Sheng et al., 2012) (Figure 4.1). At later stages of disease development once lesions have established, a central black ‘pupil’ can be observed (Goulds & Polley, 1990). Grey mycelium is often

found within the stems of colonised plants and whiteheads may appear in the severe cases (Jones et al., 1995). At the end of the season under severe epidemics, lodging occurs due to softening and rotting of the stems carrying the weight of the developing ear (Ray et al., 2006).



Figure 4-1: Eyespot disease symptom and lesions (Taken by Al-azri, 2015).

Survival of Oculimacula spp.

Oculimacula spp. can survive saprophytically for a period of three years on straw debris (Macer, 1961) (Figure 4.2). Garrett in (1975) showed that the eyespot pathogens have weak ability to compete with other saprophytes in colonizing plant material in the soil. Furthermore, weak saprophytic survival is also noted due to slow rate of straw decomposition as resting hyphae is the main survival structure (Higgins & Fitt, 1984). Moreover, it is now clear that

grasses can harbor *Oculimacula* spp. pathogenic to wheat (Hocart & McNaughton, 1994). Several grass species such as cocksfoot (*Dactylis glomerata*), couch (*Elytrigia repens*) and annual meadow grass (*Poa annua*) have been found to be a host for *Oculimacula* spp. and the pathogens have been successfully isolated from them (Lucas et al., 2000).

Inoculum production and dispersal of Oculimacula spp.

Sporulation of *Oculimacula* spp. occurs on infected crop debris remaining after harvest and the inoculum may take a form of conidia or ascospores (Figure 4.2). According to Fitt et al. (1988) *Oculimacula yallundae* reaches sporulation peak between March and April, when the optimum temperature is between 5-16°C. However, spore production decreases between June and July when temperature is above 20°C (Higgins & Fitt, 1984). In addition, water availability is necessary to facilitate sporulation. For instance, laboratory experiments in Oregon state university where infested wheat stubble was washed in running water and placed in a covered plastic container followed by incubation in the dark at 10°C, water absorption by infected debris stimulated sporulation (Rowe & Powelson, 1973). The sexual and asexual life cycle of the fungi from the previous season results in infectious ascospores or conidia, respectively, produced when temperature is above 5°C between late autumn and winter (Bateman et al., 2000).

The quantity of remaining crop residue in the soil determines the inoculum potential. In the field conidia are dispersed by rain droplets from infected straw (Figure 4.2). Spore production under light rain (<0.3 mm h⁻¹) or during dry

periods is low comparing to heavy rain (3-23 mm h⁻¹) when spores can be collected in large quantities after an initial period of wetting (Fitt & Bainbridge, 1983). A field study demonstrated the ability of *Oculimacula* spp. to spread from the inoculum source of about 1-2 m range, carried mostly by large rain droplets 400 µm in diameter (Fitt & Nijman, 1983). Although ascospores have the capacity to be dispersed by wind over longer distances, only small numbers have been collected from infected debris during rainfall (Fitt & Bainbridge, 1983).

In addition, rainfall has been found to play an important role on eyespot disease infection and spread. Above average rainfall during winter and spring was found to be strongly associated with eyespot infection (Murray et al., 1991). A later study in the Czech Republic identified that when rainfall was higher than 3mm in the number of days from October to April has an important effect on eyespot infection (Matusinsky et al., 2009). However, a study in the UK by Burnett and Hughes (2004) found contrasting results; they stated that higher rainfall between March and May influenced disease incidence, while rainfall from September to February had no effect on eyespot incidence. This difference between published literatures indicates that the effect of rainfall is still unclear and requires further research.

Infection process

The most susceptible tissue to the fungal infection by both species is the coleoptile during seedling emergence and establishment of wheat (Bateman & Taylor, 1976). The coleoptile or outer leaf sheath of the host plant is penetrated

by hyphae produced by the germinating conidia. The infection is localized at the stem base and is rarely observed above the second node on stem bases and colonization of the leaf or root tissue has not been previously reported (Cook, 1993). The two eyespot species vary in the infection process. After spore germination, *Oculimacula yallundae* grows faster than *O. aciformis* which has a slower initial growth phase (Daniels, 1993). Furthermore, for infection to occur moisture must be present. Most inoculation experiments failed without moisture (Fitt et al., 1988). Also, at the time of infection humidity is necessary for disease development. Soulie et al. (1985) specified that 80-90% of relative humidity is required to encourage the infection process, irrespective of the ambient temperature.

Environmental factors enhancing fungal sporulation on plant debris play the similar role for the initiation of plant infection. For instance, early sown crops have been shown to develop more severe eyespot disease, most likely because there is more time under favorable conditions to infect seedlings (Burnett and Hughes, 2004). An observed experiment showed that the level of eyespot in a crop established through ploughing was much higher than when the crop was established under minimum tillage (Jalaluddin & Jenkyn, 1996). This observation was supported by Burnett and Hughes (2004), who found ploughing was a factor increasing eyespot risk compared to minimum tillage. The mechanism of this effect is unclear, however it could be due to the increased populations of antagonistic organisms, or due to the ploughing of infected straw compared to being left on the surface with minimum tillage. In

addition, remaining trash may act as a physical barrier to the splash and movement of the spores.

Disease development and severity

In the UK, disease development is favored by cool damp weather in spring and mild, wet weather in winter. Hollins and Scott (1980) showed that disease incidence is determined mainly by weather conditions and although wheat plants remain susceptible to eyespot infection throughout the season the time necessary for the development of severe disease at growth stages beyond stem extension is limited. Eyespot lesions have been shown to develop within temperature range of 5 to 18°C under controlled environment and glasshouse conditions (Scott, 1971; Higgins & Fitt, 1985). Pathogen development within the stem of winter wheat in the UK in relation to thermal time was assessed by Bock et al. (2009), and the results revealed that severity of stem lesions caused by eyespot pathogens increased linearly with thermal time. The same field experiment showed that thermal time range of ca. 600-800°C days is required for the establishment of stem lesions and stem colonization and ca.1000-1200°C were needed by the both species of eyespot to reach their maximum lesion size (Bock et al., 2009).

After host penetration, *O. yallundae* infects the cell wall whereas the *O. acuformis* penetrates through the cell wall. The infection plaques of *O. acuformis* are more compact and symmetrical than the plaques of *O. yallundae*. According to Goulds and Fitt (1990), *O. acuformis* isolates develop slower on leaf sheaths and visual browning and lesions early in the season are not

apparent compared to isolates of *O. yallundae*. However, *O. acuformis* develops faster in stem tissues than *O. yallundae* (Wan et al., 2005). Yet, these differences between species become less apparent towards the end of the season (Goulds & Fitt, 1988; Ray et al., 2006). Goulds and Fitt (1990) tested eyespot species in controlled environment and in field trials and found that *O. yallundae* is more pathogenic at temperatures between 10-15°C, whilst *O. acuformis* is more pathogenic at temperatures below 7°C. Furthermore, using accumulated thermal time (degree-days) revealed that at higher temperatures the rate of leaf sheath penetration by *O. acuformis* is slower than *O. yallundae*. Wan et al. (2005) demonstrated that *O. acuformis* penetrated leaf sheaths at a significantly slower rate of 0.0067 leaf sheaths/degree-day, comparing to the penetration of *O. yallundae* that was at a rate of 0.0102 leaf sheaths/degree-day.

An inoculation experiment investigating the effect of inoculum quantity on disease severity showed disease indices were significantly higher up to GS39 in plots inoculated with *O. yallundae* than in the plots inoculated with *O. acuformis* (Burnett et al., 2012). The same experiment confirmed the consistent differences in disease development between species early in the crop growing season. In addition, eyespot disease was more severe in the 2008 season and resulted in greater yield loss due to the occurrence of lodging and whiteheads.

The severity of disease is affected by agronomic factors via influencing inoculum quantity and the time available for infection. A ten year study by Hardwick et al. (2001) showed that eyespot levels varied within years, however disease incidence was typically high, with 80-96% of infected samples each

year but with less than 1% of samples found to be severe. The trend scenario towards increased winter temperatures that was suggested by the latest predictions of climate change in the UK could encourage the predominance of *O. yallundae* over *O. acufomis* (Jenkins et al., 2010).

Yield loss caused by eyespot

Yield loss caused by eyespot disease in wheat has been found to vary depending on the severity of infection. According to Ray et al. (2006), slight disease has no effect on ear weight. However moderate or severe disease may cause up to 36% yield loss (Clarkson, 1981). Under severe epidemics yield loss can be as high as 50% (Fitt & White, 1988). Yield loss is associated with lodging at harvest (Scott & Hollins, 1974; Fitt et al., 1988). The estimated loss value from a ten year study in UK cereals caused by two species of eyespot has been shown to be 0.5% -2.2% of the total national yield (Hardwick et al., 2001). This finding was supported by a study showing that eyespot reduced national yield by almost 250,000 tons of wheat per year (Cook et al., 1991).

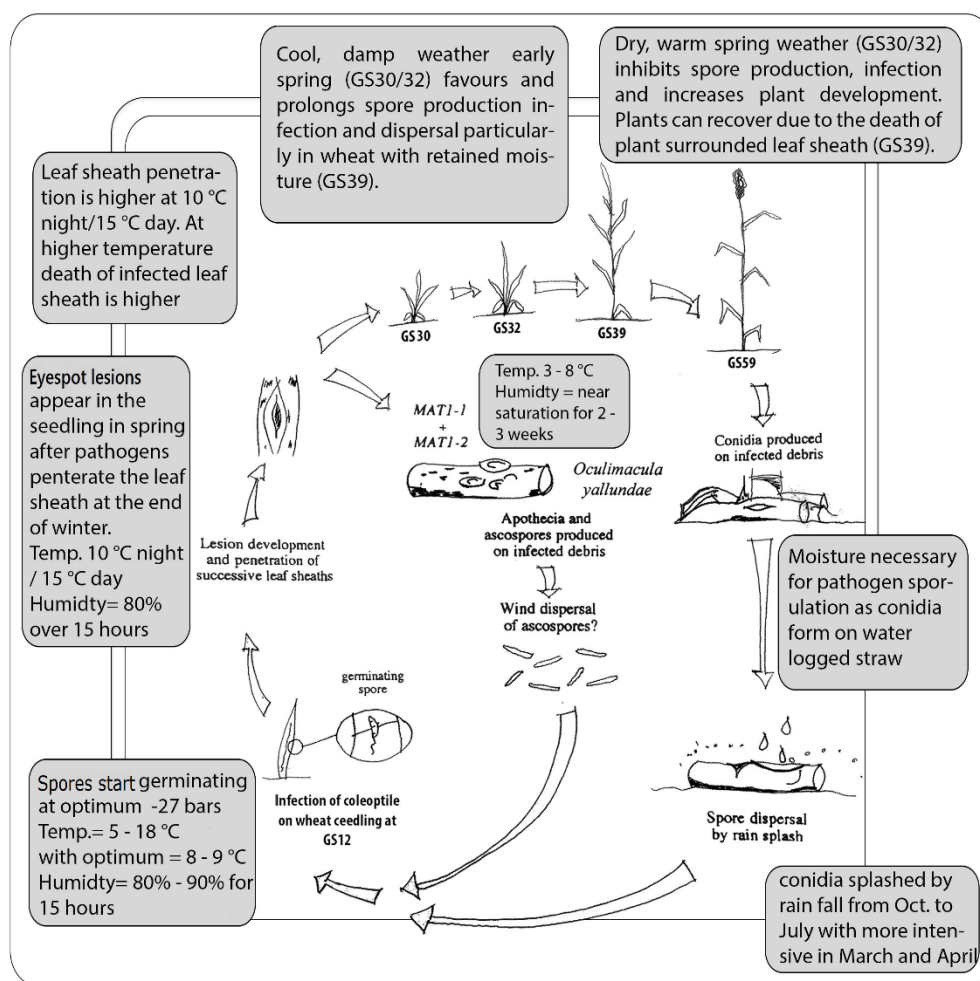


Figure 4-2: The life cycle of *Oculimacula* spp., showing asexual cycle with conidia and sexual cycle with apothecia and ascospores with climatic condition influencing its severity (Adapted from Lucas et al., 2000).

4.1.3 DISEASE CONTROL METHODS

Apart from environmental conditions, crop management has been shown to influence disease incidence. Continuous cereal rotations have been shown to increase the risk of eyespot epidemics due to survival and production of inoculum on crop debris (Colbach & Meynard, 1995). Eyespot infection could be reduced potentially and large yield savings could be achieved if a crop break

from cereals is introduced in the rotation (Cook et al., 1991). Colbach and Meynard (1995) investigated the effects of previous crop and tillage practice on eyespot incidence. They showed that when a non-host crop preceded the host crop, host residues were buried by soil inversion, however infected residues were returned to the surface when a non-host previous crop followed a host crop. This suggests that previous crop could influence eyespot in addition to inoculum levels that may be reduced for one year as a result of ploughing, but can also be returned through ploughing in the following season.

A range of soil types was found to influence eyespot disease. Light soil has been found to carry lower disease risk compared to heavy soils (Burnett, 2005). Burnett et al. (2012), suggested that clay soils are much more conducive to eyespot due to their water holding capacity that creates a suitable micro climate for the pathogen to reproduce more rapidly. Date of sowing also influences disease outcomes. For example, a higher eyespot incidence has been reported with early sown cereals (Colbach & Saur, 1998). Moreover, Gutteridge and Hornby (2003) found less eyespot infection in the spring with late sown crops. It has been suggested that the reason of a higher eyespot infection in the earlier sown crop is through extending the period for both infection and disease development (Cook et al., 1991; Colbach & Saur, 1998).

Using resistant cultivars to eyespot can be an effective method as part of control strategies against the disease. Wheat varieties may carry resistance to eyespot via the function of *Pch1* and *Pch2* (Cadle et al., 1997). It is thought that much of the UK cereal production relies on varieties that contain *Pch2*

gene and these cultivars are considered moderately resistant (Burnett & Hughes, 2004). The *Pch1* gene derived from *Aegilops ventricosa* confers more robust form of resistance. This gene was incorporated into breeding programmes and varieties producing varieties such as Hyperion and Grafton (Burnett et al., 2012). However, these resistant varieties are not widely grown by farmers due to the lack of other favourable agronomic characteristics.

Eyespot is controlled with a fungicide spray at early stem extension between growth stage (GS) 30 and 32 (Burnett et al., 1997). In the past fungicides such as benomyl and carbendazim were commonly used to treat eyespot disease. However, in 1981 this group of fungicides were reported to be ineffective in controlling the disease and isolates of both species that were resistant to benzimidazoles spread across the UK (Brown et al., 1984). Later prochloraz was reported to be cost effective and capable of reducing *O. yallundae* by 30-60% (Jones, 1994). However, with higher presence of *O. acuformis* in the early 1990s, prochloraz effectiveness decreased (Chapman et al., 2009). A study by Ray et al. (2004) investigated nine different fungicides against eyespot and found that prochloraz was not effective in controlling the *O. acuformis*. This was confirmed by a study that found *O. acuformis* had low sensitivity to prochloraz (Parnell et al., 2008).

Cyprodinil was later found to reduce eyespot by 82% (Babij et al., 2000). This active molecule was also shown to be effective in field experiments where *O. acuformis* predominated (Ray et al., 2004). The most effective and recent triazole fungicide, prothioconazole has been shown to be highly effective

against both *Oculimacula* spp. (Hollomon, 2012). Metrafenone launched in 2004 to control mildew was also found to be moderately active against eyespot at high doses (Burnett, 2005). The succinate dehydrogenase inhibitor, Boscalid in formulated mixture with epoxiconazole, was shown to give greater eyespot control than most fungicides commercially available against the disease (Leroux et al., 2007). A list of active fungicides currently used in UK crop protection against the disease is shown in Table 4.1.

Table 4-1: Active ingredients and product name of some fungicides used in UK.

Fungicide active ingredient	Product name
boscalid + epoxiconazole	Tracker
prothioconazole	Proline 275 / 250
penthiopyrad	Vertisan
bixafen + prothioconazole	Aviator 235 Xpro
epoxiconazole + fluxapyroxad	Adexar
fluxapyroxad + metconazole	Librax

4.1.4 ASSESSMENT OF THE DISEASE

Decisions to treat against eyespot are made according to an initial disease assessment carried out at GS30-32 (Jones, 1994). Visual examinations or the Polymerase Chain Reaction (PCR) are the two methods performed to identify symptoms or the presence of the fungal organisms causing the disease, respectively. Using PCR, the causal organisms in plants can be identified in the absence of visible symptoms as well as providing an opportunity to reject any

misidentification with other pathogens (Turner et al., 2001). PCR diagnostics is a useful tool to accurately identify the pathogens in the stem base complexes (Burnett et al., 2012). However, PCR does not help to determine the threshold level of eyespot treatment (Turner et al., 2001) and it is an expensive technique, which is not economically viable for growers. Visual assessment has been used to indicate eyespot severity in this study. Using visual assessment, it is possible to calculate the disease index by assessing the eyespot disease infection in the trial using one of four classes as shown in Table 4.2.

Table 4-2: Category of eyespot disease (Scott and Hollins, 1974).

Category	Disease infection symptoms
Clean	No visible symptoms
Slight	Slight eyespot (one or more small lesions occupying in total less than half of the circumference of the stem)
Moderate	Moderate eyespot (one or more lesions occupying at least half of the circumference of the stem)
Severe	Severe eyespot (stem completely girdled by lesion-tissue softened so lodging would occur)

4.1.5 EYESPOT DISEASE RISK MODELS

Eyespot disease risk assessment was developed to assist growers in making decisions for the treatment of crops against the disease. The main objective was to identify the diseased crops in need of treatment via determining a threshold level of eyespot early in the season at GS31. Weather data has also been used

to predict the threshold level of the disease; however, this was not successful due to the loss of eyespot lesions failing to penetrate the stem by shedding out outer plant leaves (Polley & Clarkson, 1978). Other threshold-based assessments have relied on assessment of the number of infected stems at the beginning of stem extension and recommending treatment if percentage stems with penetrating lesions exceeded 20% (Jones, 1994). However, this threshold method is not effective for crops that have not passed the threshold level at the start of stem extension but proceed to develop severe infection later in the season (Hughes et al., 1999). Furthermore, the forecasting and threshold methods appeared to be reasonably effective whilst eyespot disease was caused predominantly by *O. yallundae*. However, the increasing dominance of *O. aciformis* which develops slower, makes it difficult to assess visually at stem extension, making the use of forecasting and threshold less effective (Burnett et al., 2000).

The difficulties of detecting infection by *O. aciformis* before visual symptoms develop maybe overcome by using molecular diagnostic tools such as PCR (Nicholson and Turner, 2000). Moreover, accumulated degree-days could be used to produce an eyespot development scale that can differentiate between species and more accurately predict disease severity and the requirements of chemical control (Wan et al., 2005). On the other hand, development of accumulated risk score later on to predict the risk of economic damage of eyespot by Burnett and Hughes (2004) was more accurate than single threshold method. The risk assessment was based on data analysis of 341 untreated eyespot wheat crops. The crops were assessed in need of treatment based on

the level of eyespot incidence at the beginning of the season associated with economic yield loss. The six risk factors: soil type, previous crop, tillage, sowing date, eyespot at GS31-32 and March/April/May rainfall were identified by logistic regression analysis and risk point scores were weighted for each level of each factor. Burnett et al. (2012) updated this risk assessment later by predicting eyespot risk and calculating treatment cost based on the likely yield loss. This latest approach can be modified to suit different situations and can be updated with new data on yield losses of eyespot (Burnett et al., 2012).

4.2. AIM AND OBJECTIVES

The overall aim of this study was to develop an epidemiological model for eyespot disease. The main objective was to test the eyespot disease model in predicting yield loss of wheat using collected data from different wheat experiments between 2004 and 2014 in the UK.

4.3. MATERIALS AND METHODS

4.3.1 FIELD SITES AND AGRONOMY OF EXPERIMENTS ON FUNGICIDE EFFECTIVENESS AGAINST EYESPOT DISEASE IN UK

This study used historical data collected through previous research projects on fungicide efficacy against eyespot disease by the University of Nottingham, Harper Adams University, as well as The Arable Group research (TAG). Summary of all data is presented in the Table 7.1 of the Appendix. Experiments were positioned across various locations between 2004 and 2014.

Experimental field locations and their GPS coordination's are shown in Table 7.2 in the Appendix. Agronomy for all trials was as standard farm practice.

Site details including region, soil type, previous crop, tillage and sowing date were recorded in the database as known factors that influence the risk of eyespot and the final outcome of the disease as shown in Table 4.3. Regions were recorded as south, north, east and west to investigate the importance of geographical location. Tillage practice, plough or minimal cultivation was also recorded. Crop rotation was known and included in the analysis to determine the importance of host previous crop for eyespot disease. Trials covered a range of soil types including sand and clay loam soils that has also been recorded from all trials location.

Table 4-3: Agronomy factors influencing final disease outcome (from Burnett et al. 2012).

Agronomy factors	Level
Sowing date	<6 October or >6 October
Tillage	Minimum or Plough
Soil type	Light, Medium or Heavy
Previous crop	Non-host, Other cereals and Wheat.
Region	North, East, West, South

Soil K, P and Mg was analysed for each field site prior to sowing. Cultivars grown were assigned eyespot resistance score based on their ranking on the AHDB-HGCA recommended list published by the Home Grown Cereals Authority (2012). The database included trial sites, which were inoculated with

Oculimacula spp., and naturally infected presented in Table 7.3 in the Appendix. In addition, climatic data were obtained from the official weather station at Sutton Bonington campus University of Nottingham for the research sites located in Leicestershire county and met office-RAF Shawbury weather station for the Newport and Harper Adams research sites located in Shropshire county.

Moreover, fungicide treatments that have been tested during the period of 2004 to 2014 as well as field rate per hectare of each fungicide were recorded in the database that are presented in Table 7.4 of the Appendix. The main indicator of eyespot severity was disease index (DI) calculated for GS31-32, GS37-45 and GS70-80 (Zadoks et al., 1974) of wheat development (Scott & Hollins, 1974). In addition, at GS39 and GS69 pathogen DNA was extracted from plant material as described by Ray et al. (2004) and TaqMan probe quantitative Real-time assays were used to quantify the fungal biomass (Walsh et al., 2005). Grain yield was recorded and corrected to 15% moisture content.

4.3.2 CROP SAMPLING IN 2012/13 AND 2014/15 FOR CROP SIMULATION MODEL

This fieldwork was carried out at Sutton Bonington campus, University of Nottingham during winter wheat growing season for two years 2013 and 2015. In order to meet the aims of this study, the development of eyespot in the crop was assessed and associated agronomic practices such as previous crop, soil type, sowing date, sowing rate, tillage, and biomass data required for calibrating crop simulation model were collected. Collections of plant samples

in 2013 season were carried out at GS39 and GS69 only; however, in 2015 season the plots were sampled at GS31, GS39, GS61 and GS75. Using 0.25m² quadrat and avoiding the last 0.5m and the outer two rows of each plot, plants including tillers were dug out with a small amount of root kept the stem base intact for inspection. Each sample was then placed in a plastic bag and transferred immediately to the laboratory for further disease assessment and biomass measurement.

4.3.3 DISEASE ASSESSMENT

Disease assessment was performed immediately after sampling of the plots. The incidence of eyespot from 20 main tillers in a sample was calculated as the percentage of stems with visible eyespot lesions. Eyespot severity was assessed using the scale (0-3) previously described by Scott and Hollins (1974), where 0 was assigned to symptomless (clean) plants. Eyespot symptoms were scored as slight (1) when lesions covered less than half of the circumference of the stem; moderate (2) when lesions occupied more than half of the circumference of the stem or severe (3) when the lesions girdled and softened the stem. Disease index (DI, %) representing disease intensity (based on incidence and severity of the disease) was calculated using the following Equation;

$$(DI) = ((A) + (2 \times B) + (3 \times C)) / (3 \times T) \times 100. \quad \text{Equation (4.1)}$$

Where, DI = Disease index, A = (number of plants with slight symptoms), B = (number of plants with moderate symptoms), C = (number of plants with severe symptoms), T = (total number of assessed plants).

4.3.4 YIELD MEASUREMENT

Grain yield was harvested per plot and corrected to 15% moisture content. Fertile and non-fertile shoots were first separated and counted from each individual sample. Ears of fertile shoots were then separated from the straw. After that ears were threshed to separate grain and chaff. The fresh weight of grain, straw and chaff of each sample was recorded. To dry the sample, grain, straw and chaff were kept in the oven for 48 hours at 70°C. The samples were then collected from the oven and the dry weight of each of them was recorded.

4.3.5 THERMAL TIME CALCULATION

The mean daily temperature was calculated using the equation:

$$T_d = \frac{T_{dmax} + T_{dmin}}{2} \quad \text{Equation (4.2)}$$

Where T_{dmax} is the daily maximum temperature and T_{dmin} refers to the daily minimum temperature, T_d is the mean daily temperature.

Accumulated thermal time was also calculated using the method of Bock et al. (2009) using the following equation

$$TT = \sum_d T_d \quad \text{Equation (4.3)}$$

The starting point for calculating the thermal time was from sowing date to emergence with first/second leaf unfolded. The time was determined using AHDB-HGCA wheat growth guide. The date between sowing to GS11 was estimated using AHDB-HGCA depending on sowing time, if crop was sown in

September then it takes 11 days, if sowing was in October then emergence will be after 15 days. However, if sowing was in November then emergence will take 26 days. Therefore, thermal time from sowing to GS12 was accumulated when temperature between 5°C and 18°C using the Equation 2.3 if $\leq 5^{\circ}\text{C}$ and $\geq 18^{\circ}\text{C}$. Thermal time for GS31 was also accumulated between 5°C and 18°C using Equation 4.3 from sowing to GS31, if $\leq 5^{\circ}\text{C}$ and $\geq 18^{\circ}\text{C}$. However, the starting point to calculate thermal time of GS39 was the date of GS31 and it was accumulated between 8°C and 15°C to the date of GS39 using Equation 4.3 from GS31 to GS39, if $\leq 8^{\circ}\text{C}$ and $\geq 15^{\circ}\text{C}$.

4.3.6 CONCEPTUAL DISEASE MODELLING

The hypothesis of this work was that a disease model could be built to predict the disease development and yield loss in relation to crop phenology using results from previous literature on conditions favouring sporulation, infection and disease development and severity. Four disease models are used to follow the progress of disease through crop development. The infection potential model at GS12 was developed to incorporate the potential of inoculum production and quantity under different environmental and agronomy factors (Figure 4.3). Once infection occurs at GS 12/13, lesion development (Fitt et al., 1984; Bateman et al., 2000) is initiated under environmental conditions for each site until reaching GS31/32 where DI is predicted fitting with the phase of leaf sheath penetration (Scott, 1971, Higgins & Fitt, 1985).

The disease severity model at GS39 of the crop relates to lesion establishment on the true stem (Fitt et al., 1988) and environmental and agronomy parameters

together with fungicide treatment are used to predict disease severity at GS39. Harvest reduction model to predict the yield loss of the crop uses disease severity at GS39 and fungicide treatments (Ray et al., 2006).

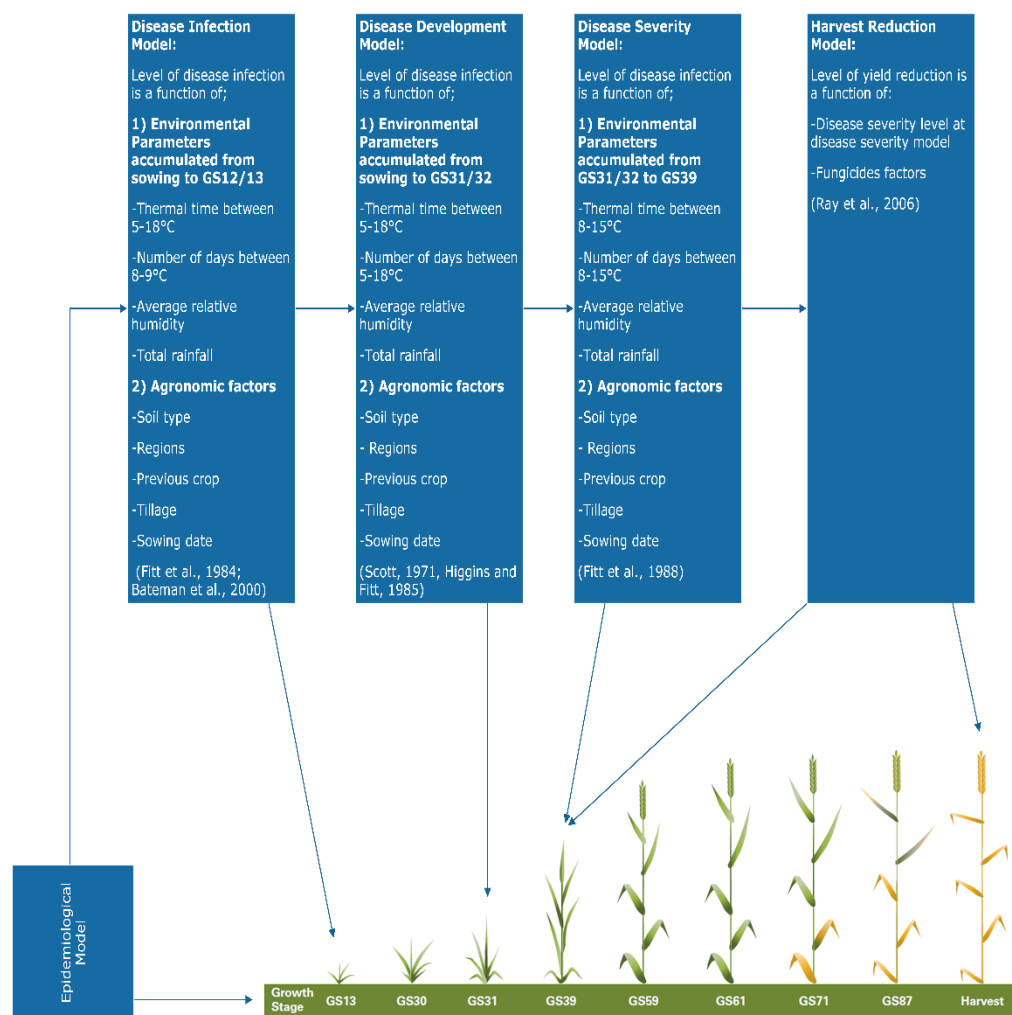


Figure 4-3: Conceptual disease model representing different stages of disease development.

4.3.6.1 INFECTION POTENTIAL MODEL (IPM)

This model builds from the data available on the environmental parameters influencing the infection potential of the disease at the beginning of the season. The interactions of thermal time, relative humidity and rainfall were assessed together with agronomic factors at GS12/13 and infection potential taken from observed data of disease incidence at GS31/32 to develop the model according to the following equation:

$$IP = f(TT_{12/13}, ND_{12/13}, RH_{12/13}, TR_{12/13}, Ag.F) \quad \text{Equation (4.4),}$$

Where Table 4.4 shows the names of the variables and the expressions used in Equation. 4.4.

Table 4-4: Variable names and the related expressions when predicting IP .

Variable name	Description
IP	Infection potential as disease incidence from GS31/GS32
$TT_{12/13}$	Thermal time between 5 °C -18 °C from sowing to GS12/13
$ND_{12/13}$	Number of days between 8 °C - 9 °C from sowing to GS12/13
$RH_{12/13}$	Average relative humidity from sowing to GS12/13
$TR_{12/13}$	Total rainfall (mm) from sowing to GS12/13
$Ag.F$	Region, Previous crop, Soil type, Tillage and Sowing date

4.3.6.2 DISEASE DEVELOPMENT MODEL (DDM)

The model is based on data available at GS31 about infection potential from GS12/13 to GS31/32. The disease development model (**DDM model**) at GS31/32 is a function of infection potential data and environmental and agronomic parameters:

$$DI = f(IP_{12/13}, TT_{31/32}, ND_{31/32}, RH_{31/32}, TR_{31/32}, Ag.F) \quad \text{Equation (4.5),}$$

Where Table 4.5 introduces the names of the variables and the expressions used in Equation 4.5.

Table 4-5: Variable names and the related expressions when predicting DI .

Variable name	Description
DI	Disease index at GS31/32
$IP_{12/13}$	Output from infection potential model at GS12/13
$TT_{31/32}$	Accumulated thermal time between 5 °C and 18 °C from sowing to GS31/32
$ND_{31/32}$	Number of days between 5 °C and 18 °C from sowing to GS31/32
$RH_{31/32}$	Average relative humidity from sowing to GS31/32
$TR_{31/32}$	Total rainfall (mm) from sowing to GS31/32
$Ag.F$	Region, Previous crop, Soil type, Tillage and Sowing date

4.3.6.3 DISEASE SEVERITY MODEL (DSM)

The disease severity model is developed by interactions between the output from disease development model and environmental factors including fungicide treatment. Here we predict the severity index SI using the following function:

$$SI = f(DI_{31/32}, TT_{39}, ND_{39}, RH_{39}, TR_{39}, Ag.F, FG) \quad \text{Equation (4.6)}$$

And Table 4.6 shows the descriptions of the variables in Equation 4.6.

Table 4-6: Variable names and the related expressions when predicting SI .

Variable name	Description
SI	Disease severity index at GS39
$DI_{31/32}$	Output from disease development model
TT_{39}	Accumulated thermal time between 8 °C - 15 °C from GS31 to GS39
ND_{39}	Number of days between 8 °C - 15 °C from GS31 to GS39
RH_{39}	Average relative humidity from GS31 to GS39
TR_{39}	Total rainfall (mm) from GS31 to GS39
$Ag.F$	Region, Previous crop, Soil type, Tillage and Sowing date
FG	Fungicide treatments

4.3.6.4 YIELD REDUCTION MODEL (YIELD LOSS)

Harvest reduction model was built by using simulated yield taken from a random simulation of data representing the same field. The model with intercept effect was included in the fungicides factors. Therefore, level of severity and yield increase or reduction at GS39 due to the specific fungicides is performed via investigating the differences in the fungicide factors. The following equation was used to build the model:

$$yield \sim Gaussian(myield, y),$$

$$Yield = f(SI39, FG) \quad \text{Equation (4.7)}$$

Table 4.7 shows the descriptions of the variables in Equation 4.7.

Table 4-7: Variable names and the related expressions when predicting yield loss.

Variable name	Description
Yield (t/ha)	Yield
SI39	Output from disease severity model
FG	Fungicide treatment

4.3.7 MODEL ESTIMATIONS

Data were analysed using regression analysis with statistical software (R 3.1.2. statistical computing and graphics) (Crawley, 2005). Fit of general linear models was assessed as the percentage variance accounted (R^2) for the disease index data (2004-2014) for eyespot from UK field trials, combined with historical climatic data.

In statistical modelling, generalised linear models (GLMs) might be used to determine whether a target variable is influenced by one or more variables using a linear additive model and a link function generalising the predicted mean (see below); more particularly in the inference of the relationship between a response variable Y (disease in our case) and a set of n independent variables (predictors) $z = (z_1, \dots, z_n)$ (environment and agronomy factors in this study) (Davison, 2003). GLMs consist of three elements:

1. A probability distribution function f from the exponential family.
2. A linear predictor $\eta = z^T \alpha$.
3. A link function g such that $E(Y) = \mu = g^{-1}(\eta)$,

Where; $E(Y)$ is the expected value of Y and μ is the mean of the distribution.

The unknown parameters α can be determined by maximum likelihood, maximum quasi likelihood or Bayesian techniques. There are several types of link function and their use depends on the type of the response data. For

example in the case of a dichotomous outcome variable, f is taken as the binomial distribution, defining the binomial family of GLMs. In this case the common choices for link functions are:

- **The canonical logit link:** $g(p) = \log(p(z))/(1 - p(z))$,
- **The probit link:** $g(p) = \phi^{-1}(p)$,
- **The complementary log-log link:** $g(p) = \log(-\log(1 - p))$.

But in case of count data such as the data used in this study, Poisson regression assumes the response variable Y has a Poisson distribution, and assumes g to be the logarithm of its expected value can be modelled by a linear combination of unknown parameters, i.e. the link function is $g(p) = \log(p(z))$. This form of Poisson regression is sometimes called a log-linear model.

Fitting the GLMs is using maximum likelihood estimation; the sample observations y_i arising from a probability density function $f(Y | \alpha)$ is known, but the vector $\alpha = (\alpha_1, \dots, \alpha_p)$ is unknown; the likelihood function is the conditional probability of observing the sample given α , which is

$$L(\alpha) = \prod_{i=1}^n f(y_i | \alpha), \quad \text{the log-likelihood function is}$$

$$\log(L(\alpha)) = \sum_{i=1}^n \log f(y_i | \alpha) \text{ and by differentiating the likelihood or the log-}$$

likelihood functions we obtain the value of α , defined as the maximum likelihood estimator $\hat{\alpha}$ of α (Harell, 2001).

Additionally, to assess the statistical significance and measures goodness of fit of the models, indicators tests like (**LR**), Akaike's Information Criterion" (**AIC**) and Bayesian Information Criterion (**BIC**) (Harell, 2001; Davison, 2003; Crawley, 2005). Moreover to determine whether candidate model parameters are statistically significant we also need to determine the goodness of fit between each incremental form of model and the measured data. To this end a number of measures are available to us:

Nagelkerke's generalised R^2

This measures the proportion of explained deviance in a model and it is defined

$$\text{by: } R_N^2 = \frac{1 - \exp(-LR / n)}{1 - \exp(-L^0 / n)},$$

Where; LR is the log-likelihood ratio test statistic and L^0 refers to the null model. It extends the definition of the proportion of explained variance R^2 used in linear models to the explained deviance in logistic models. Values of R^2 are between 0 and 1, with 0 denoting that the model does not explain any variation and 1 denoting that it perfectly explains the observed variation.

Mean squared error (MSE)

MSE is the mean of the square of the difference between the actual observations and the response predicted by the model. It is defined by

$$MSE(\hat{y}) = E(\hat{y} - y)^2.$$

4.4. RESULTS

4.4.1. EYESPOT DISEASE MODEL DEVELOPMENT

The epidemiological disease model was developed using historical data obtained from different locations in the UK (2004 - 2014) and experimental data from published literature on epidemiological parameters as shown in Table 4-8.

Table 4-8: Environmental and epidemiological parameters from previous published experiments on eyespot disease.

Epidemiological parameters	Environmental parameters	References
Inoculum production and dispersal	Temp 5-16 °C RH= near saturation	(Fitt et al., 1988, Fitt, Bainbridge, 1983)
Infection	Temp=5-18 °C RH=80-90%	(Scott, 1971; Fitt et al., 1988)
Lesion development	Temp=5-18 °C RH=80	(Scott, 1971, Higgins and Fitt, 1985)
Lesion establishment	Temp=8-15 °C RH=80	(Fitt et al., 1988)

*Disease development stages and its environmental parameters obtained from literature.

4.3.9 INFECTION POTENTIAL MODEL (IPM)

The infection potential model was fitted using the following equations:

$IP \sim \text{Poisson}(mIP)$, Therefore;

$$\log(mIP) = aTT_{12/13} + bND_{12/13} + cRH_{12/13} + dTR_{12/13} + eRegion + fPreviousCrop + gTillage + hSoilType + iSowingdate \quad \text{Equation (4.8)}$$

Where coefficients a, b, c, d, e, f, g, h, and i are related to the environmental and agronomy factors introduced in Table 4.4.

The agronomy variables used were selected from published literature on the prediction of eyespot disease (Burnett et al., 2012).

Table 4-9: Poisson regression output, showing the effect explanatory variables have upon disease infection potential at GS12/13 using data of UK inoculated winter wheat experiments obtained from different locations between 2004 and 2014.

Parameter	Coefficients	Estimate	Std. Error	Z value	Pr(> z)
	Intercept	-1.229e+01	8.865e-01	-13.859	<2e-16***
	TT12/13	1.640e-02	8.018e-04	20.458	<2e-16***
Environmental	ND12/13	-5.145e-01	5.051e-02	-10.187	<2e-16***
	RH12/13	1.448e-01	9.490e-03	15.256	<2e-16***
	TR12/13	-3.097e-02	2.783e-03	-11.127	<2e-16***
Region	West	-2.036e+01	2.1061e-01	-9.667	<2e-16***
Base=East	North	-1.518e+01	9.135e-01	16.618	<2e-16***
Soil type	Heavy	-4.023e-01	9.034e-02	-4.453	8.47e-06 ***
	Light	3.179e-01	1.490e-01	2.133	0.0329*
Base=Medium					
Previous Crop	(Continuou				
	s Wheat)	2.622e+00	1.414e-01	18.546	<2e-16***
Base=Other					
Cereal	(Winter	-1.485e-01	1.563e-01	-0.950	0.3421

	Wheat)				
	(Oil Seed	-5.215e-02	1.437e-01	-0.363	0.7166
	Rape)				
	Legumes	-2.603e+00	2.902e-01	-8.971	<2e-16***
Tillage	Minimal	1.817e+00	8.121e-02	22.371	<2e-16***
Base=plough					
Sowing date	> 6 October	-8.988e-01	1.667e-01	-5.393	6.94e-08 ***
Base=<6					

*Signifiant codes: 0 '****' 0.001 '***' 0.01 '**' 0.05 '.' 0.1 '' 1

*TT12/13= Thermal time between 5 °C -18 °C from sowing to GS12/13, ND12/13= Number of days between 8 °C - 9 °C from sowing to GS12/13, RH12/13= Average relative humidity from sowing to GS12/13, TR12/13= Total rainfall (mm) from sowing to GS12/13.

*TT & RH increasing disease infection significantly, in contrast ND & TR decreasing disease significantly.

Table 4.9 shows that thermal time and average humidity had a positive (increasing) influence on disease potential from sowing to GS12/13, while number of days when temperature is between 8°C to 9°C from sowing to GS13 and total rainfall from sowing to GS13 had a slightly negative effect (decreasing). The coefficients of these four environmental factors (thermal time, number of days between 8°C to 9°C, relative humidity and total rainfall) are highly significant (p-value= <0.001). In fact the majority of the predictors or their underlying levels have significant coefficients apart from previous crop of winter wheat and oilseed rape. The variable, number of days with temperature between 8°C and 9°C from sowing to GS13, has a coefficient of -0.5145 which means that for each single day increase in this factor, the predicted incidence of eyespot at GS12/13 decreases by 40% (as $\exp(-0.5145)=0.59779 \sim 60\%$). Similarly, the incident rate for thermal time at GS12/13 'TT12/13' is 0.01649. Therefore, IP will increase by 1.66% for every single degree-day increase in TT12/13. Total rain at GS12/13 (TR12/13)

contributes to decrease of IPM by $\exp(-0.03097) = 0.9695$. (That is 96% so a decrease of 3.05%), whilst RH contributes to increase of IPM by $\exp(0.1448) = 1.156$. Thus for any increase in total rainfall (mm) at GS12/13 there will be a reduction of 3% in prediction of the disease IP while for any % increase in average RH there will be an increase of 1.2% in prediction of IP.

The West and North regions had significantly less IP than the east region ($P < 0.001$). The model predicted the infection potential of the West region to be $\exp(-20.36) (=1.43802\text{e-}09)$ less than the infection potential of the East. While the predicted IP in the North region was $\exp(-15.18) (=2.55511\text{e-}07)$ less than the prediction of IP if it is East region. On the other hand, heavy soil was predicted to decrease IP significantly ($P < 0.001$) at GS12/13 by $\exp(-0.4023) (=0.67)$ so there was a 33% decrease of IP than if it was a medium soil. In contrast, light soil increased the infection potential significantly ($P < 0.05$) by 1.4 times greater than if it was a medium soil.

The levels of the five categorical variables used in the model were chosen to match the data for 2004-2014 created from data available from the field experiments as explained in the methodology section of this chapter. For example, five levels explain the previous crop variable and level 5 is the reference category that is named in the data “other cereals”. From this model, the estimated IP when the previous crop was ‘Winter Wheat’ would be 86.2% of that of other cereals whilst for oil seed rape it would be 94.9% of it. However, these coefficients were not significant. The predicted IP with legume as a previous crop was significantly ($P < 0.001$) (less $\exp(-2.603) = 0.074$)

producing a reduction of 92.6% in relation to prediction for other cereals. The output also indicates that the infection potential (IP) for previous crop when it is continuous wheat was 13.79 times greater than the infection potential for other cereals. Minimal tillage is associated with a significant increase in infection potential ($P < 0.001$), 6.2 times greater than with ploughing. However, sowing date after 6 October caused disease infection potential to decrease significantly ($P < 0.001$) by 60% compared to the sowing date when it is before 6 October.

As a measure of goodness of fit for the linear relationship between the predictor variables and the response variable the coefficient of determination (R^2) was used. The result for this dataset shows that 99% (adjusted R^2 , i.e. average R^2 per degree of freedom) of the variance in the response variable Infection potential (IP) would be explained by the predictor variables (agronomic and environmental factors).

4.3.10 DISEASE DEVELOPMENT MODEL (DDM)

Disease development model was fitted using the following equation:

$$DI \sim \text{Poisson}(mDI),$$

$$\log(mDI) =$$

$$IP_{12/13} + aTT_{31/32} + bND_{31/32} + cRH_{31/32} + dTR_{31/32} + eRegion +$$

$$fPreviousCrop + gTillage + hSoilType + iSowingDate \quad \text{Equation (4.9)}$$

In fact, for simplicity and as introduced previously in the **DDM model**, coefficients $IP_{12/13}$, a , b , c , d , e , f , g , h , and i are related to the variables introduced in Table 4.5.

Table 4-10: Poisson regression output, showing the effect explanatory variables have upon disease development at GS31/32 using data of UK inoculated winter wheat experiments obtained from different locations between 2004 and 2014.

Parameter	Coefficients	Estimate	Std. Error	Z value	Pr(> z)
	Intercept	-7.866e+00	1.788e+00	-4.399	1.09e-05***
Disease incidence	IP12/13	2.572e-02	1.158e-03	22.202	< 2e-16***
Environmental	TT31/32	-8.354e-04	2.042e-04	-4.092	4.28e-05***
	ND31/32	1.453e-03	1.956e-03	0.743	0.457442
	RH31/32	1.148e-01	1.934e-02	5.935	2.94e-09***
	TR31/32	3.537e-05	3.755e-04	0.094	0.924952
Region	West	-2.908e-01	8.151e-02	-3.567	0.000361***
Base= East	North	5.575e-01	1.324e-01	4.211	2.54e-05***
Soil type	Heavy	-2.274e-01	1.284e-01	-1.770	0.076677
Base= Medium	Light	-1.775e-02	1.258e-01	-0.141	0.887801
Previous crop	(Continuous wheat)	1.730e-01	3.392e-02	5.101	3.39e-07***
	(Winter wheat)	-9.530e-01	1.691e-01	-5.637	1.73e-08***
	Base=Other cereal (Oilseed rape)	-8.228e-01	1.445e-01	-5.695	1.23e-08***
	Legumes	-4.253e+00	4.138e-01	-10.278	< 2e-16***
Tillage	Minimal	3.173e-01	7.392e-02	4.292	1.77e-05***
Base= Plough	Sowing date				
	>6 October	-1.725e-01	9.332e-02	-1.849	0.064497
Base=<6 October					

*Significant codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

* $IP_{12/13}$ = Output of disease incidence from infection potential model at GS12/13, TT31/32= Thermal time between 5 °C -18 °C from sowing to GS31/32, ND31/32= Number of days between 5 °C - 18 °C from sowing to GS31/32, RH31/32= Average relative humidity from sowing to GS31/32, TR31/32= Total rainfall from sowing to GS31/32. *ND, RH & TR increasing disease infection significantly, in contrast TT decreasing disease significantly.

Table 4.10 shows that, disease infection potential predicted by IPM caused the disease index of DDM to increase significantly ($p < 0.001$). So for any unit increase in IP, the DDM disease index will increase by 2.6% in. Disease index at GS31/32 decreased slightly with greater thermal time. For a one degree-day increase in thermal time, the disease index decreased by 1%. On the other hand, average relative humidity at GS31/32 increased the disease index significantly ($P < 0.001$) by 12% ($\exp(0.11480) = 1.12$) for each unit. Although values for number of days from sowing to GS31/32 (ND31/32) and total rainfall from sowing to GS31/32 (TR31/32) were positive, they were not found to have a significant effect on disease index. Geographical location was found significant with the West associated with less severe disease by 35% than East region and North locations were expected to have a DI 1.75 times greater than in the East.

Soil type was not significant; no effect on disease development was predicted at GS31/32. All categories of previous crop reduced the disease index in comparison to 'other cereals' ($P < 0.001$) apart from 'continuous wheat' that increased the disease index significantly: 19% greater than the expected disease index for other cereals. While legumes caused the greatest disease index reduction (98.6%) followed by winter wheat (61.4%), oil seed rape showed a 56% reduction in comparison to other cereals. Minimal tillage increased disease index as it was estimated to be 1.3 times the disease index for ploughing. The sowing date variable was near significant ($P = 0.064$), so disease index could be affected by this factor, late sowing date reducing the disease index by 15%.

4.3.11 DISEASE SEVERITY MODEL (DSM)

Disease severity model was fitted using the following equation:

$$SI \sim \text{Poisson}(mSI),$$

$$\log(mSI) =$$

$$DI_{31/32} + aTT_{39} + bND_{39} + cRH_{39} + dTR_{39} + eRegion + fPreviousCrop + gTillage + hSoilType + iSowingDate + jFG \quad \text{Equation (4.10)}$$

Where coefficients of the prediction $DI_{31/32}$, a, b, c, d, e, f, g, h, i and j, were introduced preciously in Table 4.6.

Table 4-11: Poisson regression output showing the effect explanatory variables have upon disease severity index at GS39 using data of UK inoculated winter wheat obtained from different locations between 2004 and 2014.

Parameter	Coefficients	Estimate	Std. Error	z value	Pr(> z)
	Intercept	1.314e+00	1.972e-01	6.800	1.05e-11 ***
Disease	DI31/32	-2.231e-03	6.165e-04	-3.618	0.000296 ***
	TT39	-2.192e-04	4.564e-05	-4.803	1.56e-06 ***
Environmental	ND39	-1.531e-02	8.817e-04	-17.362	< 2e-16 ***
	RH39	2.412e-02	2.314e-03	10.424	< 2e-16 ***
	TR39	4.459e-03	3.096e-04	14.402	< 2e-16 ***
Region Base= East	West	6.861e-01	3.902e-02	17.583	< 2e-16***
	North	1.093e-01	4.172e-02	2.169	0.008818**
Soil type	Heavy	2.708e-01	4.057e-02	6.675	2.47e-11 ***
Base= Medium	Light	-1.374e-01	4.393e-02	-3.127	0.001766 **
Previous crop Base=Other cereal	(Continuous wheat)	1.105e-01	1.834e-02	6.023	1.71e-09 ***

	Winter wheat	-1.218e-01	4.818e-02	-2.528	0.011471 *
	Oil seed rape	-3.547e-01	5.134e-02	-6.909	4.88e-12 ***
	Legumes	5.104e-01	2.876e-02	17.745	< 2e-16 ***
Tillage Base=	Minimal	-1.444e-01	3.072e-02	-4.700	2.60e-06 ***
Plough					
Sowing date	>6 October	6.153e-01	2.368e-02	25.988	< 2e-16 ***
Base=<6					
October					
Fungicides	(bixafen and Prothioconazole)	-5.239e-01	5.252e-02	-9.976	< 2e-16 ***
	(epoxiconazole and fluxapyroxad)	-4.998e-01	6.414e-02	-7.793	6.55e-15 ***
	Base= prothioconazole	-1.242e-01	1.403e-02	-8.848	< 2e-16 ***
	epoxiconazole	-2.037e-01	1.370e-02	-14.869	< 2e-16 ***
	(boscalid and epoxiconazole) cyprodinil	-2.012e-01	1.670e-02	-12.050	< 2e-16 ***

*Significant codes: 0 **** 0.001 *** 0.01 ** 0.05 . 0.1 ' 1

* DI31/32= Output from disease development model at GS31/32, TT39= Thermal time between 8 °C -15 °C from GS31/32 to GS39, ND39= Number of days between 8 °C - 15 °C from GS31/32 to GS39, RH39= Average relative humidity from GS31/32 to GS39, TR39= Total rainfall from GS31/32 GS39. *TR & RH increasing disease infection significantly, in contrast ND & TT decreasing disease significantly.

The above Table 4-11 shows that disease severity evaluated at GS39 was reduced significantly ($P=<0.001$) by disease index observed at GS31/GS32. With any unit increase in DI31/32, the estimated SI39 from this disease severity model is seen to decrease by 0.22%. All environmental variables linked to humidity and rainfall was associated with slight disease severity increase (2.5% and 0.5% respectively) whilst thermal time and number of days between 8°C and 15°C from GS32 to GS39 are decreasing the disease severity (0.03% and 1.5% for TT and Nb of dry days, respectively).

In addition, both locations West and North increased SI significantly comparing to East, almost two times greater than east for a Western location

but only 11% increase when located in the North UK region. Heavy soil increased SI, being 1.31 times the SI for medium soil (the reference). In contrast light soil was associated with decrease in SI with almost a 13% reduction in comparison to medium soil. Interestingly, legumes despite their role as disease reduction factor in the earlier models, were increasing the disease severity index estimating an index 1.6 times the other cereal index; continuous wheat was less influential: 1.1 times the other cereals index; oilseed rape was associated with a decrease of 30% and ‘winter wheat’ a decrease of 12%.

Unlike the previous models, minimal tillage reduced SI in DSM by almost 13.5% from ploughing, whilst sowing the crop after 6th October was associated with an increase in SI by 85% in comparison to sowing before the 6th of October. All fungicide treatments which were applied at GS31/32 had a significant effect of reducing SI with bixafen and prothioconazole being the most effective by reducing SI at around 41% than if just epoxiconazole was used. Cyprodinil was least effective associated with reduction of 18% compared to the reference of epoxiconazole.

4.3.12 YIELD REDUCTION MODEL (YIELD LOSS)

Yield reduction model was fitted using the following equation:

$$yield \sim \text{Gaussian}(myield, y),$$

$$\log(myield) = SI_{39} + FG \quad \text{Equation (4.11)}$$

Where, *myield* is the yield normal growth and the coefficients of the prediction **SI₃₉** and **FG** were introduced in Table 4.12.

Table 4-12: Gaussian regression output, showing the effect of explanatory variables on yield using trial data of inoculated winter wheat obtained from different locations between 2004 and 2014.

Parameter	Coefficients	Estimate	Std. Error	t value	Pr(> t)
	Intercept	10.59824	0.17678	59.952	<2e-16***
Disease	SI39	-0.02232	0.00241	-9.259	<2e-16***
Fungicides	prothioconazole	0.26639	0.12932	2.060	0.0397*
	(boscalid and	-0.14230	0.13017	-1.093	0.2746
	Base=epoxiconazole	0.03257	0.13017	-1.093	0.2746

*Signifiant codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

*SI39= Output from disease severity model, Fungicides= different fungicides factors. * Disease severity at GS39 decreasing yield slightly, however only prothiconazole chemical increasing yield significantly.

Environmental variables were not significant to the 95% confidence level and were removed from the final model presented in Table 4.12. As the frequency of some fungicides was less than 10 they were also removed from the log-regression analysis. The above results showed that, SI at DSM is reducing the yield but only by 2.2% for each unit increase in SI. Only prothioconazole fungicides showed a difference with the reference (epoxiconazole) with a 30% increase in yield.

4.3.13 YIELD LOSS SCENARIO USING ESTIMATED PARAMETERS

Disease development and yield loss caused by disease can be predicted by changing multiple variables from the previous models (Table 4-13).

Table 4-13: Disease development and yield loss prediction performed using multiple variables in the model.

Model	<i>mIP12/13</i>	<i>mDI31/32</i>	<i>mSI39</i>	YLM*
Observed data	8.13*	0.92	56	9.4
Scenario 1	6.0	1.5	46	9.5
Scenario 2	87	4.1	71	9.0
Scenario 3	14.1	3.0	82	8.8

* Observed data of IP12/13 taken from GS31/32 observed data

* Yield loss depends only on SI39 model.

In Table 4-13, the Observed data constitutes the reference scenario with IP, DI, SI and yield resulting observation made under the conditions of sowing date before 6 October in East region with a medium soil and where the previous crop was oil seed rape under minimal tillage, with measured environmental variables: TT12/13=152.8, ND12/13=0, RH12/13=80.2 and TR12/13=19.4.

Scenario 1- changing TT to be two times greater (=305.6 at GS12/13) and ND also to be two times greater (=2 at GS12/13) with TR to be three times greater (=58.2 at GS12/13), keeping all other factors the same. With scenario 1, Infection Potential would be reduced by 25% (IP estimated at 6). Scenario 2 is the same as scenario 1 but with previous crop changed to continuous wheat, IP is now 14 times greater (87). Scenario 3 is scenario 2 but with minimal tillage

changed to ploughing, thus the prediction of IP in scenario 3 now nearly doubles to 14.1. This result indicates that environmental variables as well as some key agronomical factors such as previous crop and tillage play an important role in influencing the potential disease infection at GS12/13. Continuous wheat as previous crop increases the disease risk; however, ploughing reduces the disease risk, whilst increasing environmental factors increasing rate of crop development seem to decrease the disease risk as evaluated by the infection potential.

Applying the scenarios with an estimated IP to the disease development and severity models is shown in Table 2-13. At GS31/32, observed data was TT31/32=1162.5, ND31/32=129.8, average RH31/32=91.1 and TR31/32=337.0 to be TT =2300, ND=128, same RH and TR=1000. DI will slightly increase to 1.5. However, when keeping all factors the same changing previous crop from oil seed rape to continuous wheat, DI will be four times greater than the observed as scenario 2 of DDM shows in Table 2-13, while if minimal tillage is replaced with ploughing DI in scenario 3 will decrease to 3%. Therefore, the worst scenarios that increase the disease risk is previous crop as continuous wheat.

At GS39, the worst scenario in DSM was when minimal tillage changed to ploughing that increased SI from 56% observed data to 82% at scenario 3, unlike IPM and DSM as showing in Table 4-13. While the best scenario for lower disease is when changing TT, ND to double and TR to 3 times as SI decreased by almost 10%. The yield loss depends mainly on the disease

severity model at GS39 and as results indicated in Table 4-13, yield increased slightly by 0.1 t/ha^{-1} when SI decreased by 10% in scenario 1. However, when SI at DSM increased by 15% yield decreased by 0.4 t/ha^{-1} as in scenario 2. Moreover, yield reduction was 0.6 t/ha^{-1} when SI increased by 26% as shown in scenario 3.

4.5. DISCUSSION

The objective of this study was to develop a conceptual eyespot disease model predicting yield loss of wheat in the UK. The relative influence of the factors, region, soil type, previous crop, tillage, sowing date, weather and fungicides were examined. Historical data of surveyed sites in different parts of the UK and detailed fungicide trials were investigated to determine the influence on eyespot development and build separate disease models. Disease progress was assessed visually through sample collection, from 2004 until 2014.

Analysis of the data sets showed that climatic conditions and agronomic factors influenced disease development either positively or negatively in all models. Using Poisson regression to predict eyespot disease incidence based on different environmental and agronomy predictors accordingly, three different models were developed, IPM, DDM and DSM. These statistical sub-models were aligned with crop development. In the literature, no direct relationship was found between the incidence of plants that developed eyespot and the weekly mean value of temperature and rainfall (Fehrmann & Schrodter, 1971). They concluded that correlation coefficients were optimal for infection when

assessing whether the incidence of eyespot development was related to the environmental factors.

In this investigation, the disease model at three different stages revealed that accumulated thermal time influenced disease infection at GS12/13 significantly causing incidence to increase slightly. However greater thermal time caused slight reduction in DI at DDM and SI at DSM. This is most likely related to effects also on crop development not just on pathogen activity. It has been shown previously that pathogen activity increased with increasing temperature (Scott, 1971). Temperature above 5°C was optimal for eyespot development (Matusinsky et al., 2009). Fitt (1985) showed that the rate of penetration by *O. yallundae* increased above temperature of 6°C, which would fit with our DSM. Such a result indicates that thermal time is a good measure to assess development of monocyclic disease like eyespot (Lovell et al., 2004). In addition, relative humidity was found to be positively influencing the disease with significant from infection at GS12/13 to severity at GS39. This agrees with a study demonstrating that *Oculimacula* spores were produced abundantly on residue or even on the soil surface under warm, damp conditions (Jordan & Hutcheon, 2003). Also Murray et al. (2009) demonstrated that disease development was influenced by wet, damp conditions over winter.

The number of days when temperature was between 8°C and 9°C reduced disease infection potential at GS12/13 and disease severity at GS39 significantly. Total rainfall significantly decreased disease infection at GS12/13, but it has no effect on disease development at GS31/32. But it caused

disease severity to increase significantly at GS39. This result agrees with findings from Burnett and Hughes (2004) that higher rainfall between March and May influenced disease incidence, while rainfall from September to February had no effect on eyespot incidence. On the hand, under a changing scenario when environmental factors such as TT and number of days were set to be double and total rainfall to triple the amount of the observed. We noted that environmental factors caused the disease infection at GS12/13 and disease severity at GS39 to decrease slightly, while little increase was seen in disease development at GS31/32. This is likely to be related to positive effects of these environmental parameters on the rate of crop development, which may have negative impact on rate of eyespot leaf sheath penetration and lesion establishment on wheat stems.

Effect of regions on eyespot disease was considered with largest differences exerted in the West and North on this study. The conditions under both regions at GS12/13 significantly reduced disease infection and West conditions also reduced disease development at GS31/32 but both regions influenced disease severity at GS 39 to increase significantly. This result agreed with Burnett and Hughes (2004) who reported that location played a large role on increasing disease risk but it should also be considered with other factors including soil and weather. Heavy soil type was found in this study to decrease disease infection significantly whilst light soil increased disease infection. The role of soil type in disease severity changed at DSM, where heavy soil increased SI significantly. At DDM both types of soil had no effect on disease development. In the literature light soil has been found to carry lower disease risk compared

to heavy soils (Burnett, 2005). In fact, heavy soils are those with a large component of clay in them and greater water holding capacity as well as being characterised by small pores, which can retain water in their profile much longer than soils with large pores (Balck et al., 1970). This may explain why heavy soil influenced the disease at severity stage as soil moisture can have a significant impact on lesion development and establishment of eyespot on wheat as demonstrated by Fitt (1985). This was also noted by Burnett et al. (2012), who suggested that clay soils are much more conducive to eyespot due to their water holding capacity that creates a suitable microclimate for the pathogen.

A rotation effect was also considered within the models as a factor affecting disease index. Continuous wheat was found to have the largest significant positive effect on disease index in all models comparing it with all other crops in rotation. However, legumes reduced disease index significantly in both infection and development models whilst winter wheat and oilseed rape had no significant effects at infection model, though significantly reduced the disease than other cereal at DDM and DSM. Such a finding is supported by a study, which stated that rotation with host crop was found to be the largest cultural factor affecting disease incidence (Colbach et al., 1999). It was also found that eyespot was more if wheat was rotated with cereal crop while rotation with non-cereal crop was found to reduce eyespot incidence significantly (Cook et al., 1991).

This result may indicate that crop rotation plays an important role in inoculum production and disease development more than disease severity. In this study using multiple parameters to test the effect of disease development and yield loss under changing variables. The result showed that replacing oilseed rape with continuous wheat was increasing disease risk at IPM and DDM. Also residue of previous wheat allows plant pathogen inoculum to build up and that enhances disease infection in the next wheat crop (Fitt et al., 1990).

Minimal tillage was found to contribute significantly to higher infection potential and disease development but not to severity at GS39 where minimal tillage reduced disease significantly in comparison to ploughing. This result agreed with Suffert and Sache (2011), who found eyespot caused significant infection in tillers if straw was completely incorporated prior to drilling. However, cultivation techniques need to be used together with crop rotation to be most effective, for instance two years old infected wheat straw can be brought back to the surface by ploughing (Coalbach, 1999). In this study minimal tillage has been replaced with ploughing under different scenarios and it showed that ploughing causes higher disease risk at DSM and also reduced yield. This agreed with other risk assessment by Burnett and Hughes (2004), where ploughing was found to have greater levels of disease compared to minimal tillage.

Date of sowing also influences disease outcomes significantly at IPM and DSM. Sowing date after 6 October was found to reduce disease significantly in the disease infection model, with slight reduction in disease development

model. In contrast, sowing after 6 October influenced the disease severity to increase significantly in the DSM model. This result agreed with Yarham (1986) who found eyespot is less likely to be severe in late sown crops. Also with Fitt et al. (1990) have shown the severity of the eyespot infection in late sown crop is less due to the lower plant density. Another study has shown similar results that demonstrate that a greater eyespot incidence has been observed in early sown crop due to more accumulated temperature and extensive tiller and large canopy (Smiley, 2009).

Generally, the majority of the agronomy and fungicide predictors or their underlying levels had significant coefficients. The application of all fungicides at GS31/32 reduced disease significantly at GS39 in comparison to epoxiconazole alone-based fungicide. These results are also consistent with previous report on the efficacy of fungicide treatments against eyespot disease. Fungicides effective against eyespot disease are well discussed in the literature (Cook, 1980; Ray et al., 2004). The lack of effectiveness of epoxiconazole was expected, as all other chemical treatments assessed were known to have a certain degree of activity against eyespot. A study by Ray et al. (2004) to assess the effect of fungicides against eyespot found Opus to have poor activity against eyespot, the fungicide reduced disease index by only 2.6%. Burnett and Hughes (2004) classified epoxiconazole as similar to the untreated category when assessing eyespot chemical control. In France, the poor control by triazole fungicides against eyespot has been noted in particular against *O. acufomis* (Leroux, 1998).

Other chemicals were found here to cause significant reduction in disease severity with cyprodinil (Unix) causing the lowest reduction. Although epoxiconazole alone is not effective against eyespot, mixing epoxiconazole with boscalid (Tracker) was found to be very effective to control eyespot (O'Sullivan et al., 2007), which is in agreement with this study. Disease severity at GS 39 decreased yield only slightly by 2.2% per SI unit, whilst only prothioconazole increased yield significantly with almost 30% yield increase so on average in our data 2 t/ha. A success of fungicides to increase yield is well noted in previous studies (Ray et al., 2004). Application of fungicides in winter wheat in Southern Sweden was found to increase yield with a response of 0.3 t/ha (Wilk, 2009). Also, prothioconazole (Proline 275) was very successful in increasing yield and reducing eyespot index as demonstrated by Burnett (2005). This chemical has been considered as one of the more effective triazole fungicides and has been shown to be more effective than a mixture of Unix and Opus (Jorgensen, 2008; Burnett and Hughes, 2004). This fungicide operates by inhibiting the mycelium of the fungus and its mode of action is very similar to that of other fungicides that act as demethylation inhibitors (Baur & Schmitt, 2004).

Overall this study has shown that the majority of the weather parameters, agronomy and fungicides predictors or their underlying levels significantly influenced the disease outcome. The significant correlation result between yield loss and eyespot severity at GS39 found in this study is contrasted with no consistent correlation between yield loss and eyespot severity result found by Burnett et al. (2012). This indicated the need of further research to

investigate the role of eyespot disease on yield. Reduction of disease under scenario of increasing environmental factors in this study implies that under future climate change an increase in parameters like temperature and rainfall might decrease the distribution and severity of eyespot.

Chapter 5

5. CROP GROWTH SIMULATION USING APSIM WITH EYESPOT DISEASE MODELLING FOR WINTER WHEAT IN UK

5.1. INTRODUCTION

Crop scientists are facing several challenges to tackle increased frequency of weather extremes and uncertainty of climate change (Semenov et al., 2011). Therefore, continuous research is needed to stabilise crop yields despite extreme adverse weather due to climate change. Crop simulation models capable of addressing complex relationships can be used to better understand crop development and yield losses associated with climate variability or biotic stress. However, it is important to test and validate model performance under local conditions to account for specific environment crop interactions (Moore et al., 2014).

Modelling approaches for yield prediction have been developed worldwide for specific crops under changing environments. Agricultural Production Systems Simulator (APSIM) is a modular framework that simulates various crops including wheat and pasture production systems, soil, water and nutrient flow and their interactions with climatic conditions (McCown, 1996). It has resulted from a combination of two model approaches including Productivity, Erosion and Runoff Functions to Evaluate Conservation Techniques (PERFECT) (Littleboy et al., 1992) and a cropping system model for operational research (APSIM) (McCown & Williams, 1989). PERFECT was utilized to combine the existing crop with enhanced soil management, soil water movement and erosion. While APSIM implemented the computer simulation model of the growth, development, and yield of spring and winter wheat (CERES-wheat) model as a crop template to achieve high sensitivity of crop growth, soil water

and soil nitrogen (McCown et al., 1996). Adoption of a Common Modelling Protocol (CMP) within APSIM framework has simplified APSIM enough to allow integration with sub-models to simulate more complex agricultural systems (Moore et al., 2007).

5.2. OVERVIEW OF APSIM

APSIM consists of three models categorised as plant, environment and management. The models simulate wheat growth under various soil water and nitrogen conditions, surface residues and fertilizer applications (Holzworth et al., 2014). Thus APSIM simulates crop growth and development, under different soil characteristics and management options including various cropping systems. Wheat crop growth and development in a daily time step on an area basis (per square meter) thus simulating plant populations, rather than individual plants. The input variables required by APSIM include environmental conditions, soil characteristics, wheat cultivar information and management data (Figure 5.1).

Growth and development of wheat in this model are dependent on environment (temperature, humidity, rainfall and solar radiation), plant available water and available nitrogen in the soil. Daily meteorological data including temperature, a met file interacting with individual models within APSIM provides rainfall and radiation. The daily uptake of the soil water and nitrogen by the crop model is fed to the Soil-Wat model (water balance model that distributes water throughout the soil profile) and to the Soil-N model (nitrogen model that balances available soil carbon and nitrogen as well as their dynamics) on a

daily basis. Crop cover data is provided to the Soil-Wat model to calculate runoff and evaporation rates. At crop maturity, yield is simulated as the output of the model.

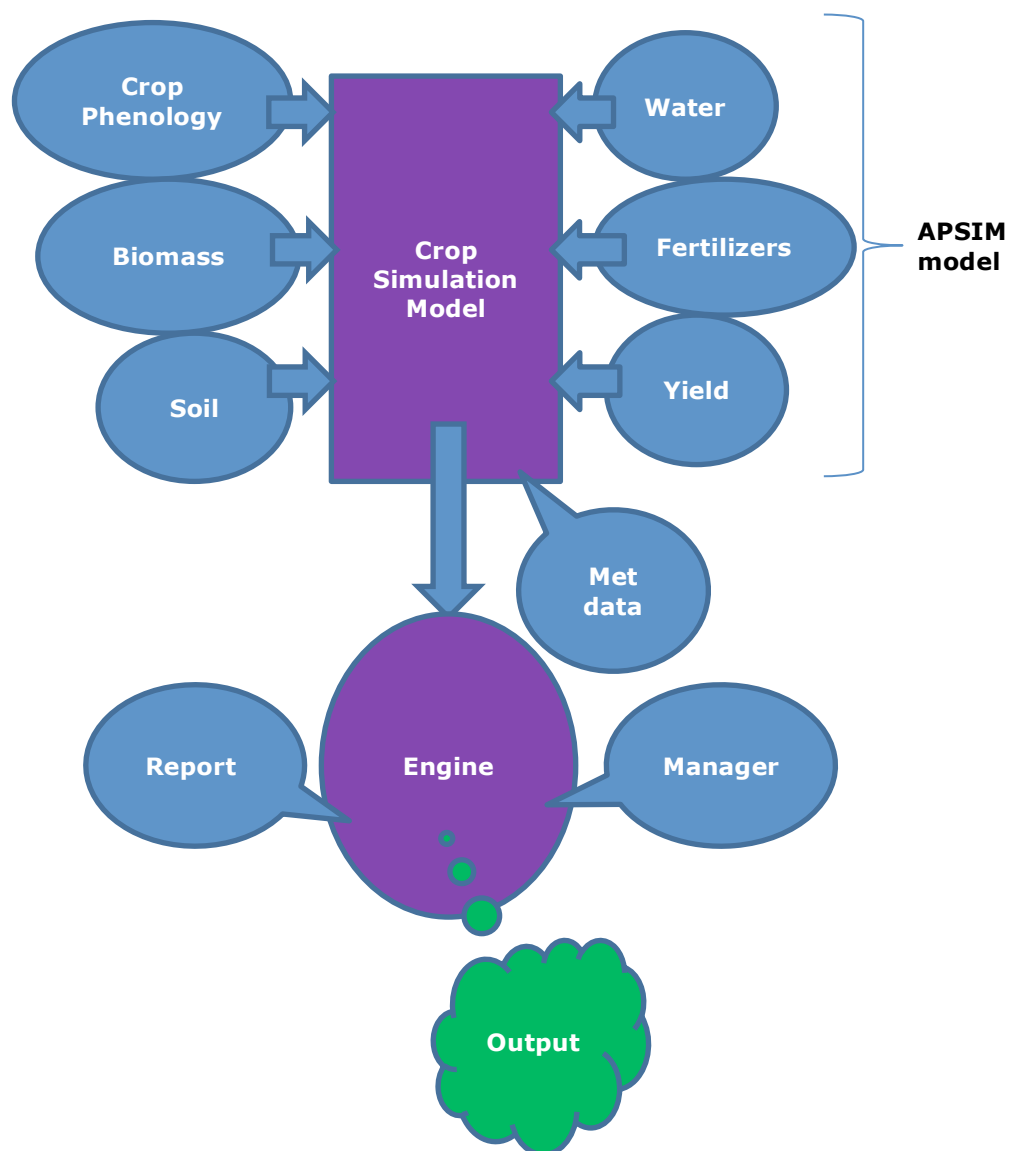


Figure 5-1: Structure of the epidemiological disease model and crop simulation model (Al-Azri et al., 2014).

5.3. LINKING DISEASE AND CROP SIMULATION MODELS

APSIM is a software tool that enables sub-models to be implemented to simulate diverse agricultural systems. APSIM was originally designed for research of the dry-land cropping systems in Australia. However, it is now used for simulating other more complex systems investigating for example resource competition between different organisms. For instance, GRAZPLAN (Moore et al., 1991) for pastures and animal production was integrated with APSIM to simulate farming systems in the Mediterranean and Temperate regions of Australia. In addition, the pasture model Grass production (GRASP) (McKeon et al., 1990) was linked with APSIM for use in the Subtropics and Tropic areas of Australia. Simulation with APSIM has provided some benefit to the agriculture production not only in Australia but also in other countries. For example, APSIM-ORYZA (Gaydon et al., 2012), was tested against diverse, replicated experimental datasets for rice-based cropping systems, different soil types, management practices, crop species, varieties and sequences representing three different countries (Australia, Indonesia and Philippines). The integrated model was able to simulate rice grain yields even in multi-season crop sequences.

One obvious disadvantage of APSIM is that it does not take into account biotic effects on final yield. Few attempts have been made to quantify biotic constraints on crop yields using the link between a crop simulation model and a biotic constraint model. One of the attempts was to integrate weed seed bank model VensimTM with APSIM to examine farm management strategies to

reduce weed seeds (Smith et al., 2005). The weed resistance in Australian rain-fed farming system was successfully investigated using the Vensim-APSIM seed bank model. However, the restriction number of weed cohorts available in VensimTM prevented the model from simulating all possible weed cohorts (Thornby & Walker, 2009; Thornby et al., 2010).

One of the approaches taken to combine conceptually and technically APSIM crop growth with disease model was DYMEX-APSIM link. A DYMEX (Computer software that allows the user to build and run computer models which describe the lifecycles and management of biological organisms) (Sutherst & Maywald, 1998) was constructed to simulate the interactions between stripe rust caused by *Puccinia striiformis* and wheat (White et al., 2004). This model was tested with APSIM to simulate four years of stripe rust trials in Wagga Wagga and Yanco, NSW Australia. The primary link between DYMEX and APSIM was leaf area index (LAI), APSIM provided LAI from the wheat model and DYMEX calculated the reduction in the green leaf area caused by disease, which was then returned back to APSIM. Thus the reduction of LAI received by APSIM allowed daily adjustment in green leaf area value and increased leaf senescence value (Which et al., 2015). Although the DYMEX rust model had limited ability to predict the proportion of disease in all years examined it demonstrated successful combination between APSIM crop model and disease model with the ability to change wheat development in response to the rust disease progress.

The performance of APSIM to simulate above- and below-ground growth, grain yield, water and N uptake, soil water and soil N of wheat crops has been previously evaluated in a temperate climate in the Netherlands (Asseng et al., 2000). The overall simulation showed good consistency where high yields of the long-term experiment were achieved, but overestimated lower yields. APSIM was chosen for the purpose of this study over the Sirius model (Jamieson et al., 1998), because of two important reasons; i) APSIM can simulate wheat growth and yields under any crop growing conditions and ii) the multi-point features within APSIM that allows it to simultaneously simulate multiple points in space and the interactions between them as well as the input and output features that simplified communication between multiple models which does not exists in the Sirius model. To our knowledge there is no previous record in the literature on the use of APSIM within the wheat farming systems in the United Kingdom. Since crop simulations require calibration and validation under local conditions, the performance of APSIM model to simulate wheat growth under UK conditions was first carried out in this study using wheat data from field experiments at Sutton Bonington, UK. To facilitate disease model implementation with crop yield simulation R scripts were executed within the framework of APSIM.

5.4. AIMS AND OBJECTIVES

The overall aim of this chapter was to evaluate APSIM for its ability to simulate winter wheat development and yield reduction due to eyespot under UK conditions.

The main objective was to implement conceptually and computationally the epidemiological disease model within a crop simulation to predict yield loss of wheat associated with eyespot disease in the UK.

This chapter comprises of two main areas of work. First, APSIM was evaluated for its ability to simulate the growth, development and yield of UK winter wheat. Second, the eyespot disease model developed in this study (see chapter 2) was implemented with APSIM to predict the effect of disease on wheat growth and yield.

5.5. MATERIALS AND METHODS

5.5.1 EXPERIMENTAL SITES AND BIOMASS DATA

Sequential growth analysis was performed on different wheat cultivars in 2012/13 and 2014/15 at the University of Nottingham, Sutton Bonington Campus (52°N, 1°W) (Table 5-1). Analysis was carried out at GS39 and GS69 in the first year and at GS31, GS39, GS61 and GS75 in the second year. Five plants at random were taken from the sampled plants per quadrat (0.25 m²) to assess the number of fertile shoots per plant. The strongest, tallest and thickest shoot of each of the five plants was then selected and the flag leaf height was measured (the start point at the base and end point is at the flag leaf), as well as length and width of the flag leaf also measured. The roots of all sampled plants within the quadrat (0.25 square meter) were then removed and total fresh weight of the plants was recorded. Then, 10% subsample of the plants were taken from the total fresh weight to do further growth analysis. The flag, second and remaining leaves of the 10% plants were then collected separately

and fresh weight of each separate set of leaves was recorded. In addition, the total fresh weight of the stems was also recorded. At GS61 and GS75 wheat ears were collected separately and their fresh weight was measured. The total green area of flag, second and remaining leaves as well as stem and ears were measured using LI-3100C area meter manufactured by LI-COR Biosciences, USA. Samples of flag, second and remaining leaves as well as stems and ears were dried in an oven at 70°C for 48 hours to obtain biomass for individual fractions.

Table 5-1 : Details of the experimental datasets used to validate APSIM for UK wheat

	Soil texture	Sowing date	Harvest year	Treatment	Data
Experiment 1	Clay loam	03/10/12	2013	Seed rates: 250 seeds m ⁻² . N applied twice and fungicides applied at three different stages.	LAI, biomass and yield
Experiment 2	Sandy loam	15/10/14	2015	Seed rates: 300 seeds m ⁻² . N applied three times and fungicides applied at four different	LAI, biomass and yield

5.5.2 METHODS OF APSIM CALIBRATION AND VALIDATION

The simulation of the winter wheat phenology, biomass, and yield was calibrated and validated using APSIM 7.5 with field measurements from the two experiments carried out in 2012/13 and 2014/15. Daily meteorological data of minimum and maximum temperature, rainfall, solar radiation, and humidity

was collected from an on-site automated weather station. Management details of the crop including sowing date, location, fertilizer applications and yield were collected as explained in methodology section in chapter 4. The biomass data required to create the calibration file was carried out as described in section 5.5.1.

The soil description (e.g. soil texture) and initial values in the APSIM soil model were parameterised with measured characteristics of the experimental field soil in the 0-30 and 30-90 cm horizons (texture, bulk density, and water holding capacity at 0.33 and 15 bar). Soil characteristics below 90 cm were not available, and were therefore assumed to be as 30-90 cm horizons. In addition, the parameter ‘crop lower limit’ for wheat in APSIM was not available for the experimental soil, and was assumed to be equal to LL15 (permanent wilting point). Moreover, the standard values for long season wheat used in APSIM that represent the maximum rate of water extraction defined for each soil layer were used to run the simulation (Table 5-2). Soil nitrogen in APSIM was initialised by soil mineral nitrogen measured at the beginning of each field experiment. Nitrogen fertiliser was applied in the simulation according to the field records for the individual experiment.

The coefficients for the genotype of the specific cultivar used in the field experiment were not available in the model genotypes list. Previously no UK winter wheat varieties have been parameterised in APSIM. Thus single winter wheat cv. Claire and the basic data of the cultivar coefficients available in the model were used to simulate APSIM. However, few alterations were made to

the phenology. Some parameters of the leaf size were adjusted based on measurements for longer wheat season in New Zealand. Tiller number was also modified for higher sowing densities. Grain number was also increased and to raise harvest index to highest possible level, together with the amount of stem mass that can be retranslated. Adjustment of the cultivar coefficients was carried out until measured values were simulated within the main growth and phenology of the crop. To analyse the model sensitivity and improve the coefficients, observed simulations were made for the growth development parameters.

Table 5-2: Experimental soil parameters used in APSIM.

Depth (cm)	Bulk density (g cm ⁻³)	Air dry (mm mm ⁻¹)	LL15 (mm mm ⁻¹)	DUL (mm mm ⁻¹)	SAT (mm mm ⁻¹)	Wheat LL (mm mm ⁻¹)	Wheat PAWC (mm) [#]	Wheat 'kl' (mm mm ⁻¹ d ⁻¹)
Clay loam								
0-15	1.16	0.10	0.312	0.380	0.531	0.312	10.2	0.06
15-30	1.16	0.18	0.312	0.380	0.531	0.312	10.2	0.06
30-60	1.18	0.18	0.330	0.437	0.525	0.330	32.1	0.04
60-90	1.18	0.24	0.342	0.476	0.525	0.342	40.2	0.04
90-120	1.20	0.24	0.342	0.484	0.517	0.342	40.2	0.03
120-150	1.20	0.24	0.342	0.484	0.517	0.342	40.2	0.02
150-180	1.20	0.24	0.342	0.484	0.517	0.342	40.2	0.02

[#]mm equivalent rainfall DUL = Drained Upper Limit, SAT = Saturation point, LL15 = water content at 15 bar, PAWC = Plant Available Water Capacity, 'kl' = the maximum daily rate of soil water extraction.

To link the disease model and the crop simulation model, the eyespot disease model was implemented within the APSIM framework. This technically integrated approach was chosen because this workflow uses BPMN standard (Business Process modelling Notation, bpmn.org), which is a standard method of expressing the workflow. Moreover, it enables the crop simulation start and

end at multiple points and allows better interaction between the models. The workflow starts with the simulation start button and it finishes at the red button as demonstrated in Figure 5-2. It follows the conceptual approach described in chapter 4.

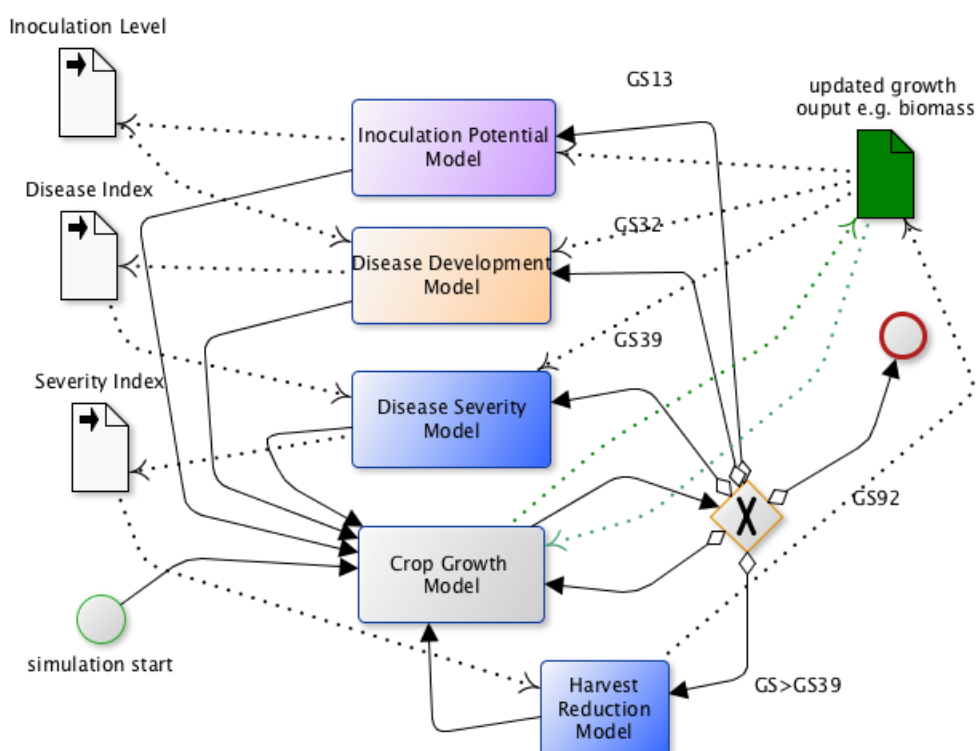


Figure 5-2: Eyespot disease crop growth workflow integration (Al-Azri et al., 2014).

5.5.3 INTEGRATING EYESPOT DISEASE MODEL WITH APSIM

Eyespot disease model was built in R script and APSIM framework allowed running R script within its framework. Therefore, disease model components were executed with APSIM simulations. Information is then passed between the disease model, which accepts data from APSIM during simulation of crop

biomass and yield and feeds back disease data such as disease index to the other models in the workflows (Figure 5.4). Some modifications were required to enable the disease model to run within APSIM framework. Average relative humidity is one of the environmental parameters used to build the epidemiological eyespot disease model. APSIM climate model did not provide this value; therefore, APSIM climate model was modified to include this additional variable.

The workflow presented in Figure 5.4 shows the implementation of APSIM with the individual disease models of eyespot developed in Chapter 4. Crop biomass is updated and yield reduced due to the disease at each step of the growth simulation orchestrated by APSIM. The first solution explored computationally was to wrap each of the models, IPM, DDM, DSM and APSIM as separate processing that can be called from within a machine-readable version as shown in Figure 5.4. This is possible using the BPMN standard for workflow and an enabling software tool. For example, the e-GRASP (Leibovici et al., 2017) offers this possibility and uses web services to wrap the processing tasks (OGC WPS: <http://www.opengeospatial.org>). Instead of using the e-GRASP workflow capability the second option leading to a similar computational organisation was to use the quasi-workflow capability within APSIM. APSIM can manage update of inputs in different management file after each time step and run a script, which can be written in R as well. That script was written to call successively the different models when the GS stage was attained as shown in Figure 5.2. Nonetheless, the two workflow approaches are based on updating directly the biomass and yield produced after

each time step in ASPIM. So first we wanted to manipulate biomass and yield but unfortunately these two variables are not ‘settable’ in APSIM. Therefore, we could not alter these variables and feed the loop in APSIM.

To overcome this obstacle, the simulation of IPM, DDM, and DSM disease model was to implement the impact directly on the components of the crop using specific models, e.g. tiller reduction, reduction due to whiteheads and reduction due to lodging models as showed in Figure 5-3. These added extra sub-models (Figure 5-4) could now generate updates on variables that can be updated in the APSIM scripting part. This offers a more realistic integration, however the added extra model (tillers, white heads and lodging reduction models) required experimental specific data to be updated to the IPM, DDM and DSM. Since this additional data of stem lodging and white heads were not available, only rough approximations were performed.

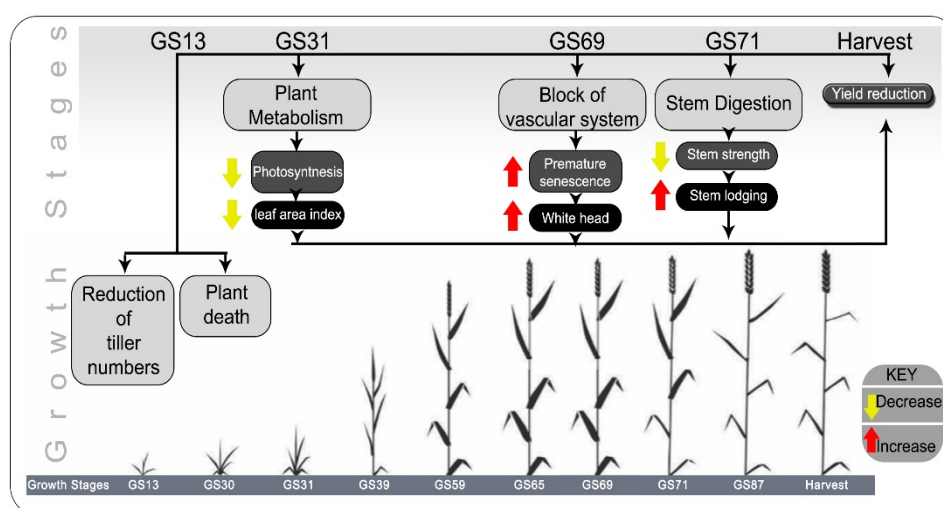


Figure 5-3: Growth stages of the wheat crop and those of *Oculimacula* spp., on different stages from GS12/13 to the harvest (Al-Azri et al., 2014).

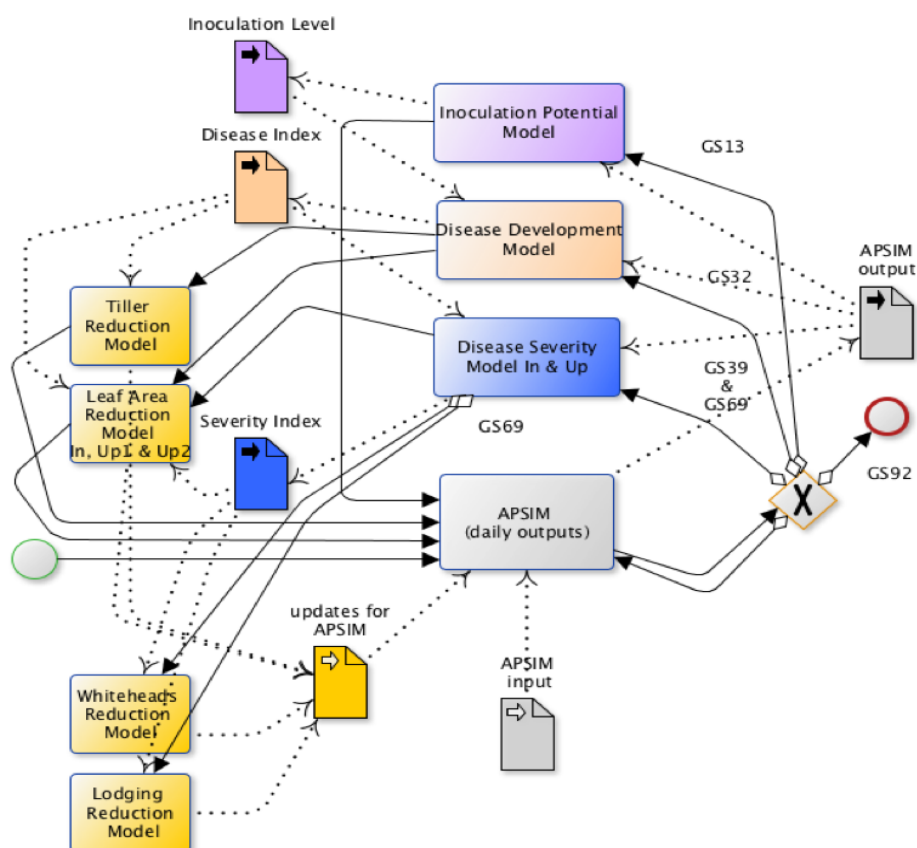


Figure 5-4: Mechanistic diagram of APSIM crop model incorporated with epidemiological disease and yield reduction model in relation to crop growth stages (Al-Azri et al., 2014).

Further step has been taken trying to calculate yield reduction due to disease within disease-APSIM model simulation. We tried to use a co-efficient, with help from the APSIM developer (Neil I. Huth). Three different manager folders were created within APSIM including kill crop, kill factor and grain kill fraction as presented in Figure 5-5. In the kill crop disease file, tillers of the crop were reduced and therefore plant population was reduced by 1% per day over a two weeks period, the script is shown in Figure 5-6.

Since the crop in the model can compensate with new tillers as the simulation proceeds final yield loss will not be overly sensitive to tiller mortality unless of

high disease severity. Whilst KL factor (rate of maximum daily water uptake per day) this used to limit the amount of water available to crop from the soil on any day in APSIM simulation. The disease script was written as shown in Figure 5-7 with a fraction value between 0.5 and 1. The disease script shown in Figure 5-8 was written for the grain. In order to reduce the grain number a killing fraction value between 0.1 and 0.01 has been set in the script so that yield directly reduced.

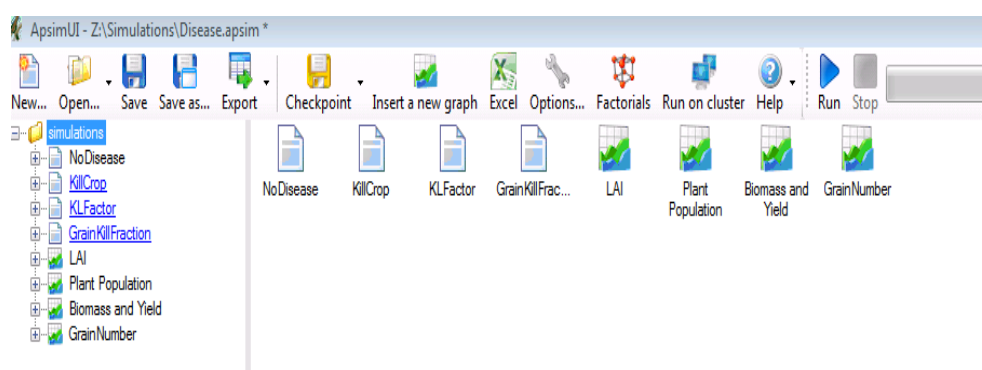


Figure 5-5: APSIM script with different disease files.

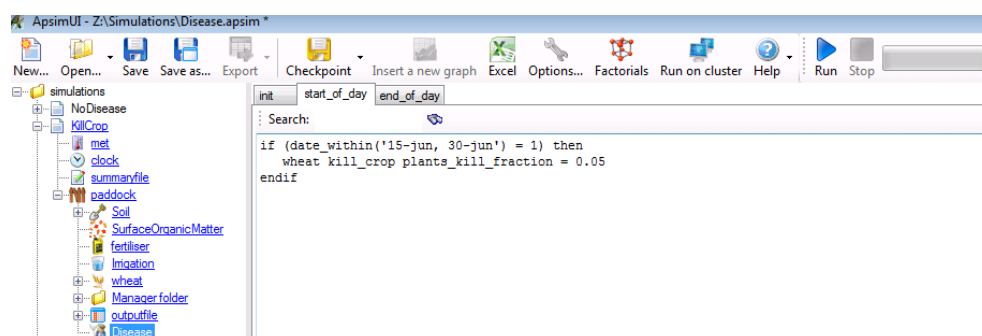


Figure 5-6: Script of kill crop disease file in APSIM.

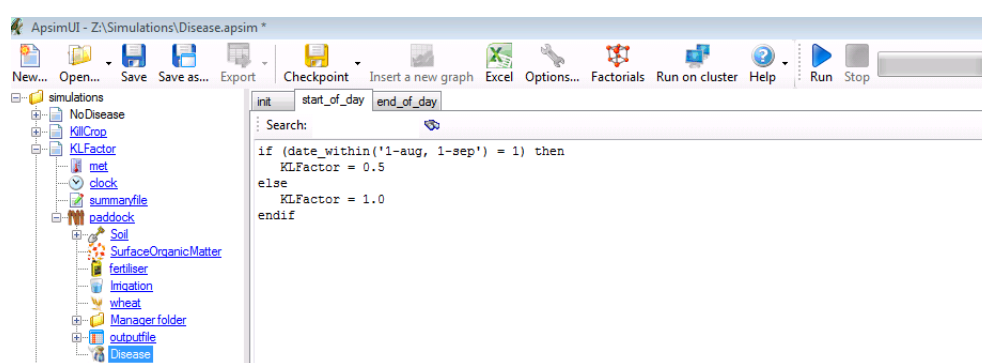


Figure 5-7: Script of KL factor disease in APSIM.

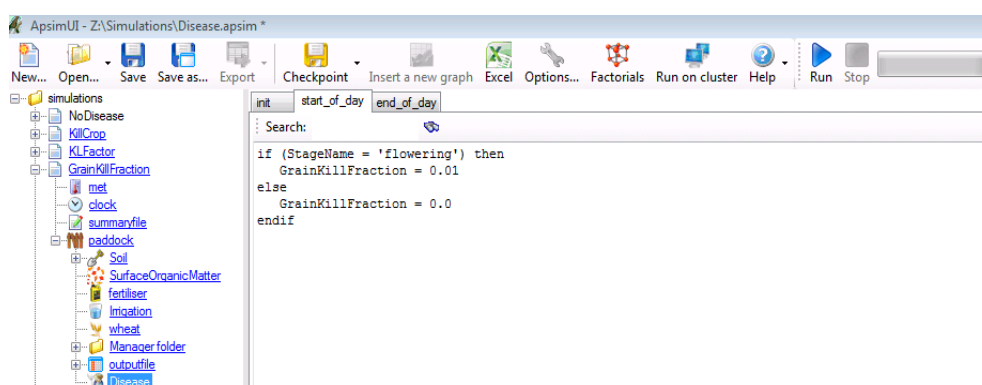


Figure 5-8: Script of Grain kill fraction in APSIM.

5.6. RESULTS

5.6.1 APSIM CALIBRATION AND VALIDATION

APSIM was used to calibrate and validate field measurements from the two experiments carried out in 2012/13 and 2014/15 at Sutton Bonington at the University of Nottingham. Generally, APSIM either over predicted or under predicted the phenology. In 2012/13 experiments there were 2 observed

phenology times at GS39 and GS69. APSIM simulated GS39 8 days earlier than observed while it simulated GS69 10 days later than observed. However, in 2014/15 where we had measurements of three phenology stages GS31, GS39 and GS69, APSIM simulation was also not very close with observed data. The simulation of GS31 was 17 days later than the observed. Whilst GS39 and GS69 were simulated 5 days and 10 days, respectively earlier than observed. The duration of thermal time from anthesis to the start of grain filling, which is one of the phenology phases in APSIM, is assumed to be 120°C day, obtained from CERES-wheat during its development. This may explain the differences between simulated and the observed phenology, which is due to the cultivar differences used in this study and in CERES-Wheat.

On the other hand, the biomass of observed data was lower than the simulated values in both experimental years. The predicted biomass of 2012/13 and 2014/15 is shown in Figure 5-9 and Figure 5-10 respectively. APSIM was unable to accurately simulate leaf area index (LAI) in both years. The predicted values of the leaf area index (LAI) were overestimated by the model at most growth stages of Oakley variety (Figure 5-11 & 5-12), particularly at the end of the season. However, LAI was predicted well with Cashel variety. Generally, APSIM prediction of the phenology, LAI and biomass was not accurate. This result led to the conclusion that APSIM model is not suitable to simulate wheat growth and development under the temperate regions of the UK.

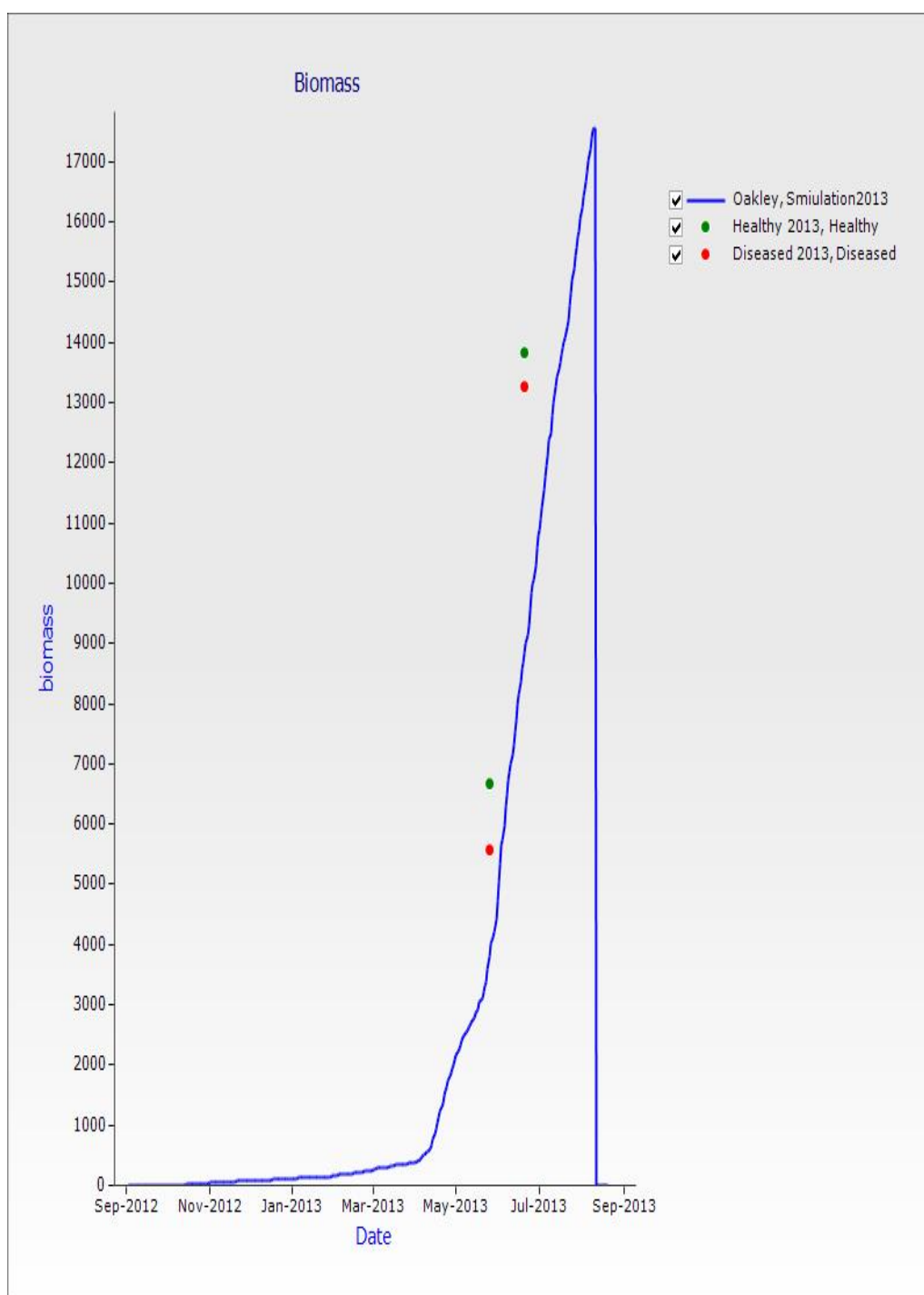


Figure 5-9: Model performance for total above ground biomass against healthy and diseased biomass using measurements data 2012/13 of Oakley variety. APSIM overestimated biomass than observed data of healthy and diseased biomass of the Oakley variety.

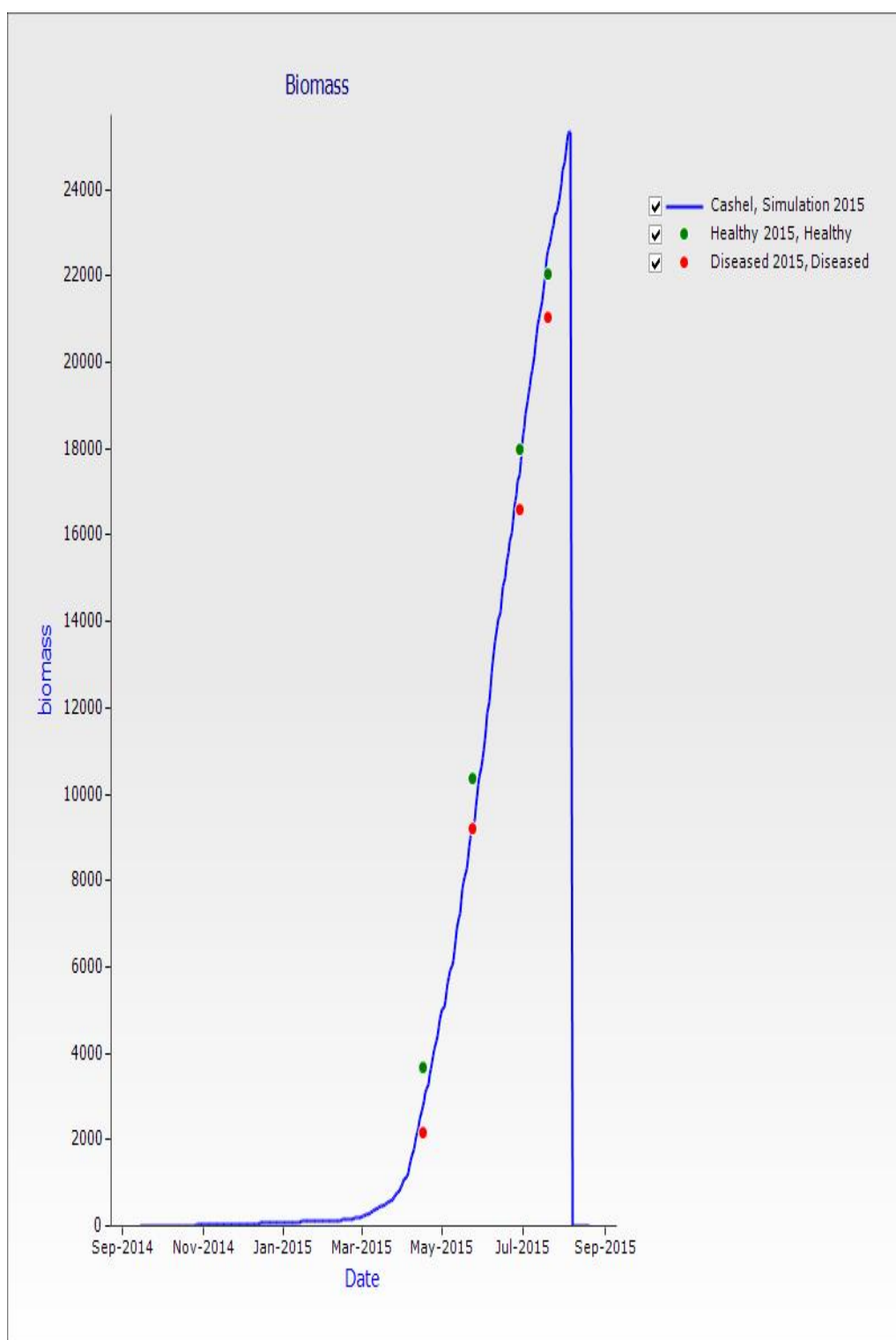


Figure 5-10: Model performance for total above ground biomass against healthy and diseased biomass using measurements data 2014/15 of Cashel variety. APSIM overestimated biomass than observed data of healthy and diseased biomass of the Cashel variety.

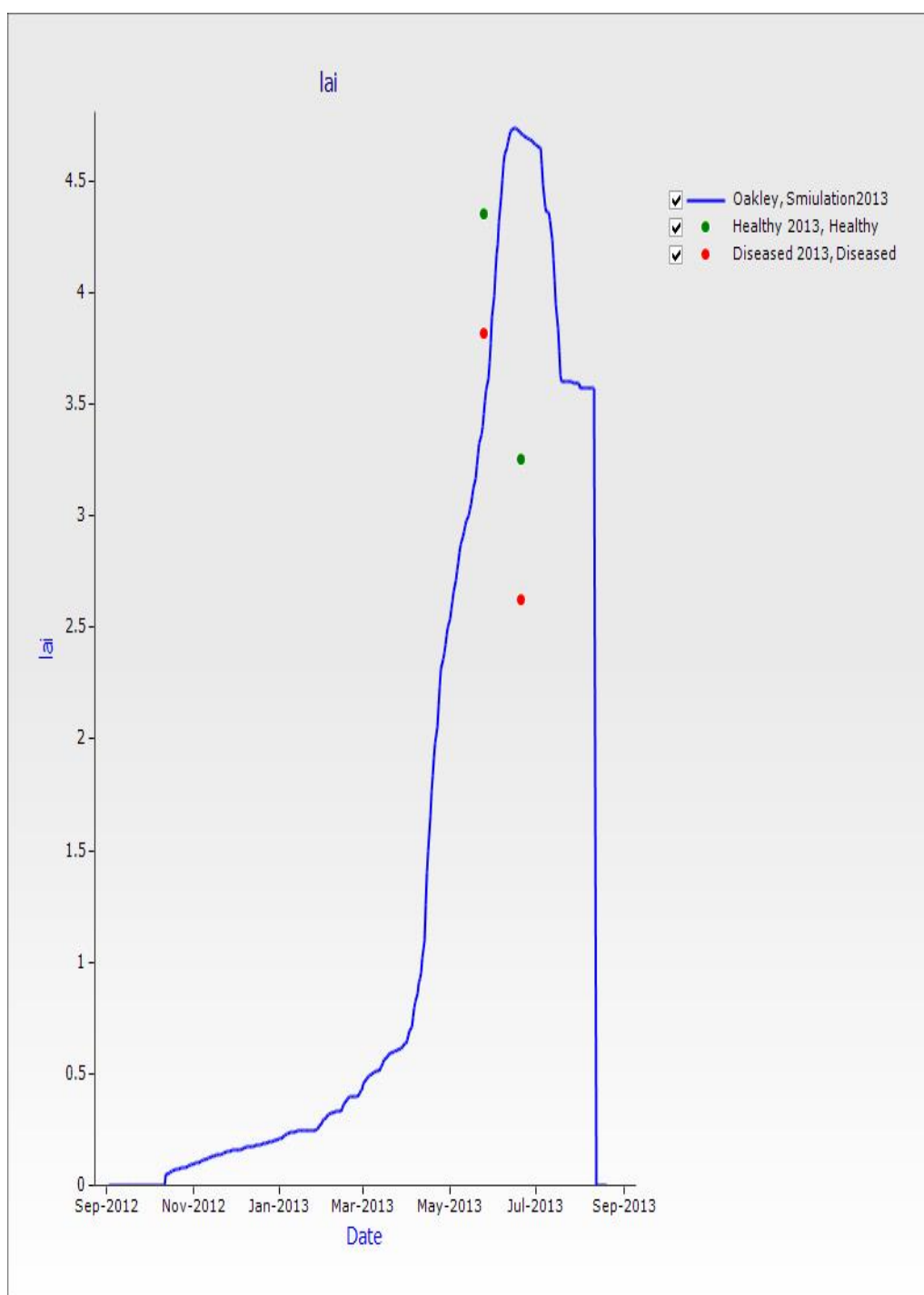


Figure 5-11: Model performance for LAI against healthy and diseased LAI using observed data 2012/13 of Oakley variety. APSIM overestimated LAI than observed data of healthy and diseased LAI of the Oakley variety.

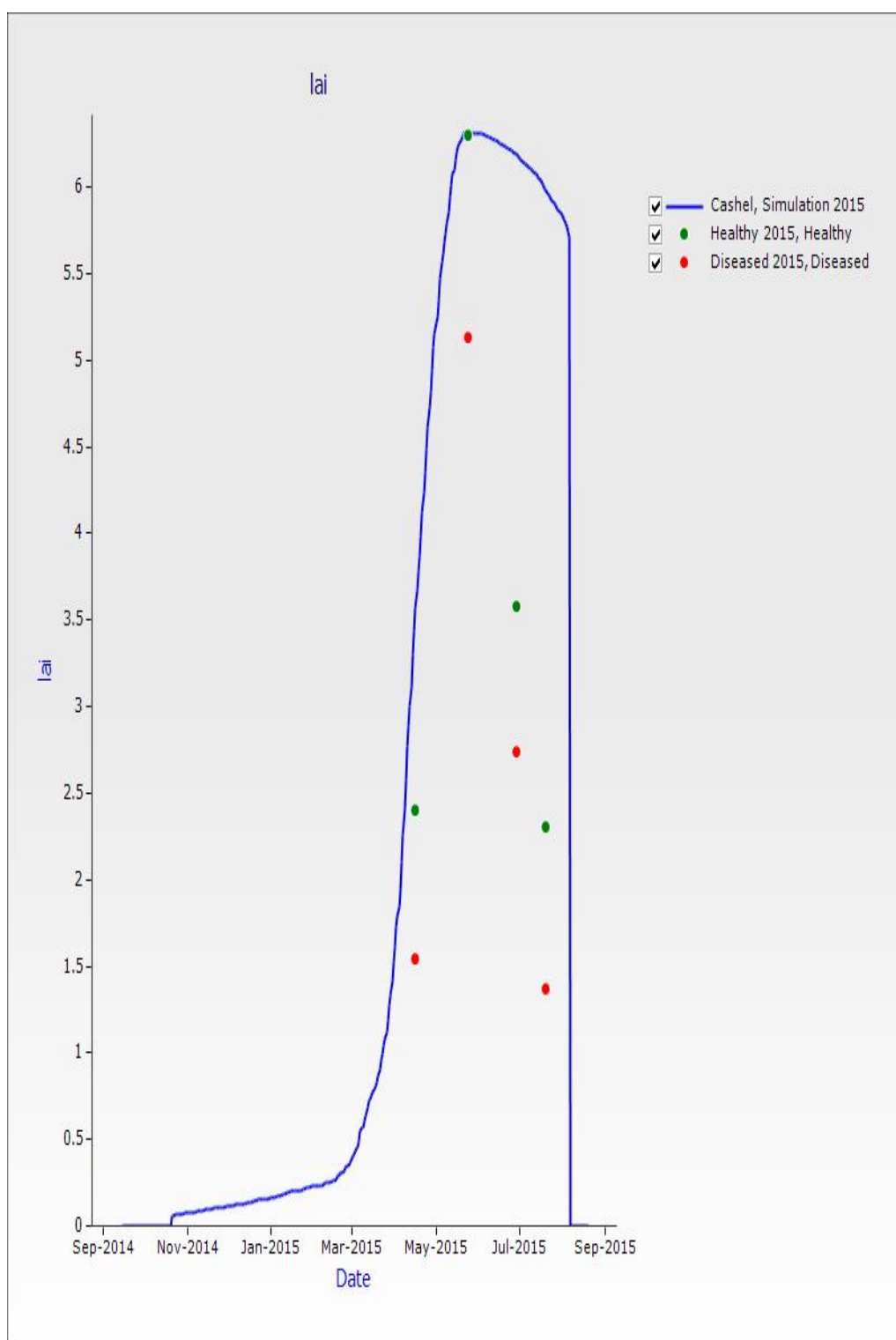


Figure 5-12: Model performance for LAI against healthy and diseased LAI using observed data 2014/15 of Cashel variety. APSIM predicated LAI close to the observed data of healthy and diseased LAI of the Cashel variety.

APSIM estimated yield accurately in 2012/13 data, the system produce a wheat yield of 7.4 t/ha, the same as observed value obtained for the healthy and 6.5 t/ha for diseased observation, indicating a good agreement between measured and predicted values (Figure 5-13). This was not the case with data of 2014/15 where APSIM under predicted the yield (Figure 5-14). The estimated yield by APSIM was almost 11 t/ha that was the same as diseased measured data; whilst almost 2 t/ha less than that of healthy yield. Overall, APSIM cannot accurately simulate yield of different wheat varieties for UK conditions.

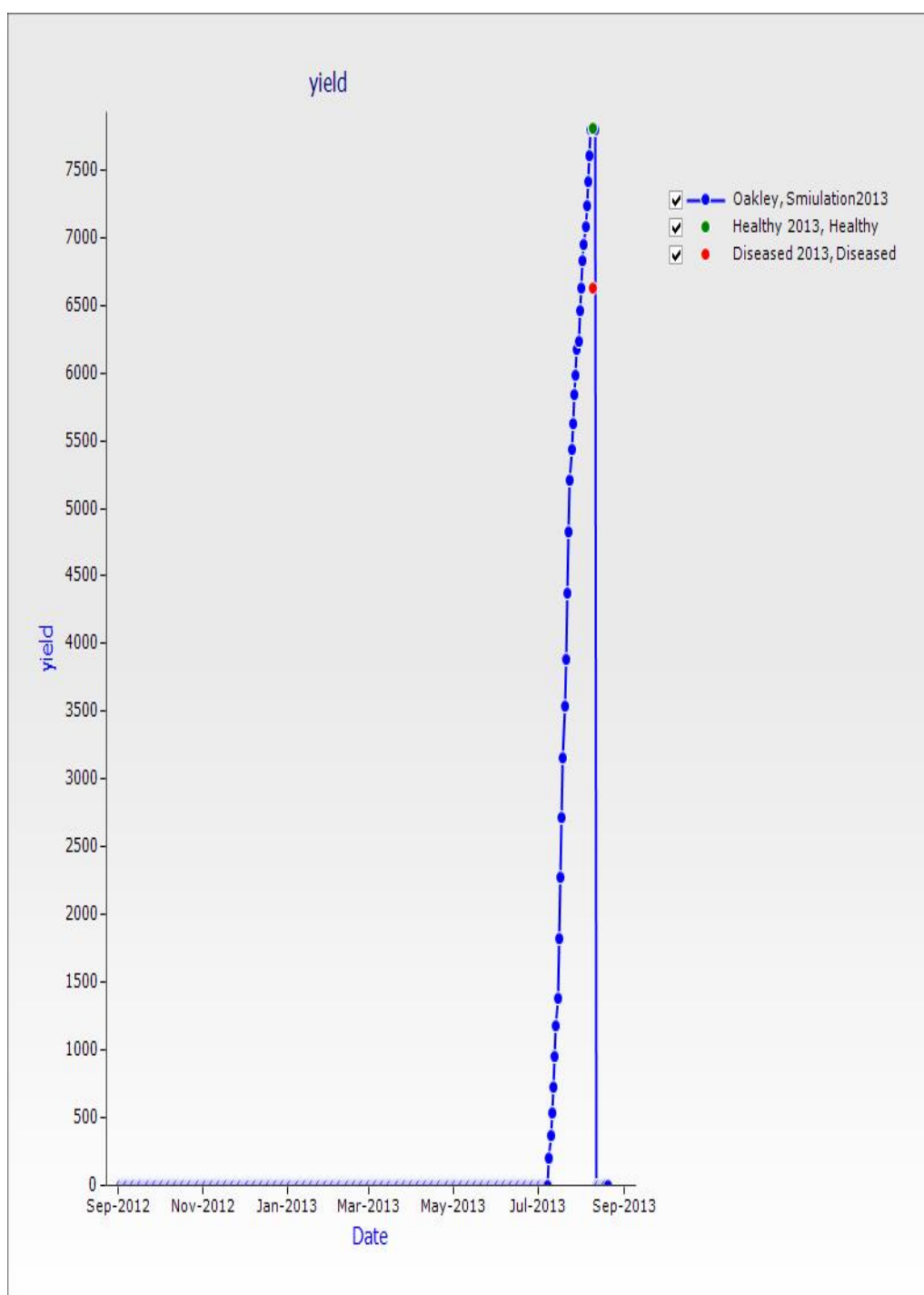


Figure 5-13: Model performance for yield against healthy and diseased yield using 2012/13 observed data of Oakley variety. APSIM Predicated yield accurately as observed data of healthy and diseased yield of the Oakley variety.

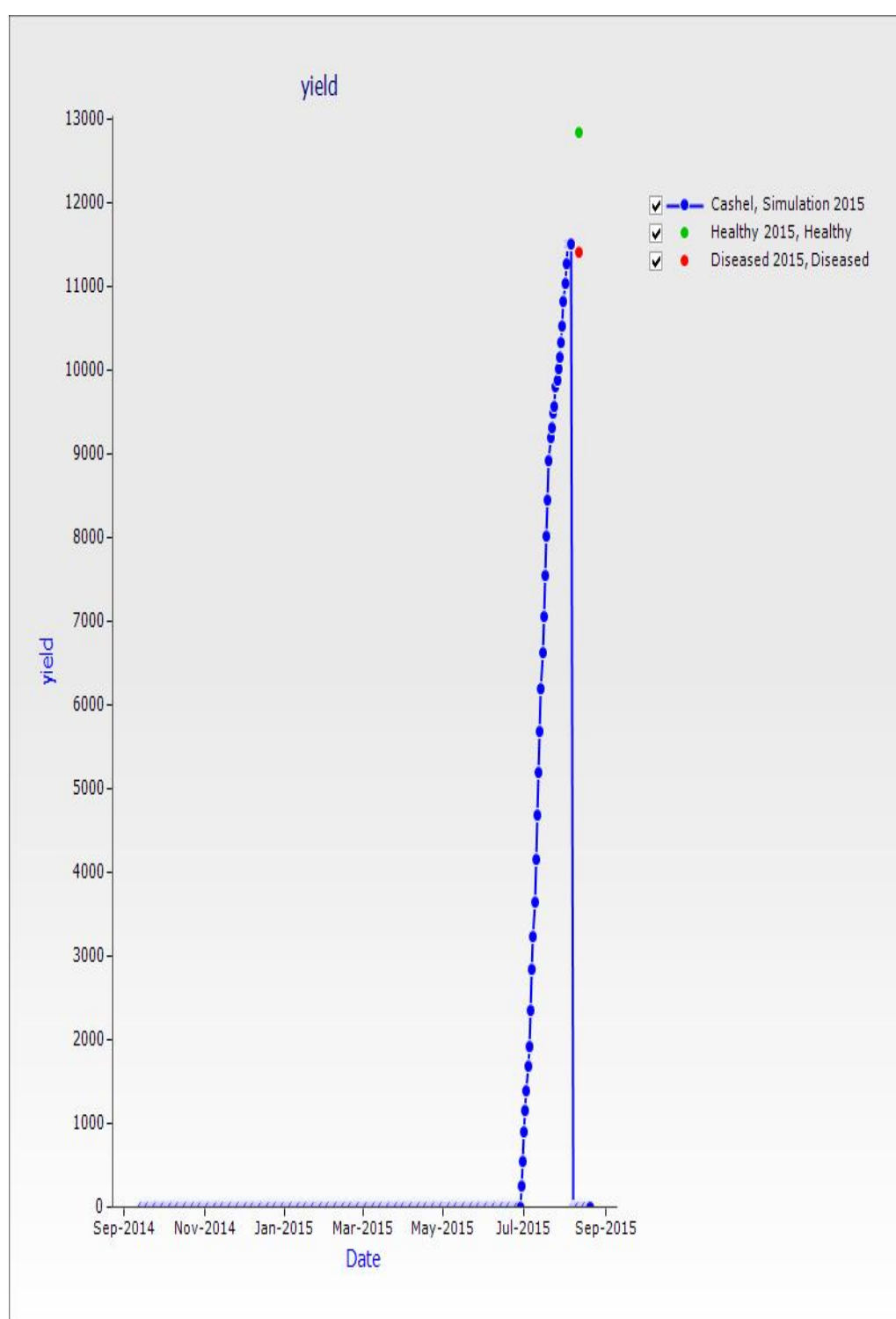


Figure 5-14: Model performance for yield against healthy and diseased yield using 2014/15 observed data of Cashel variety. APSIM underestimated yield then the observed data of healthy yield of the Cashel variety.

5.6.2 DISEASE MODELS LINKED WITH APSIM

Eyespot disease models developed in chapter 4 of this study were linked with APSIM to simulate the loss caused by disease during growth stages and final yield. The R^2 deviance for IPM, DDM and DSM models was between 0.93-0.99. IPM prediction was 9.6% disease incidence at GS13, 1.3% disease index from DDM at GS32 and 12% disease index from DSM at GS39. However, we could not alter biomass and yield to feed the loop in APSIM, therefore yield reduction model could not be implemented with disease models and as a result yield reduction cannot be simulated.

Moreover, even after adding extra models to the workflow such as tiller and white heads reduction models still yield loss could not be generated using APSIM due to the shortage of specific data needed for the models. In addition, different disease files were implemented in APSIM where specific input values reduce either the number of growing points, water uptake or grains that then reduce the yield directly. However due to inconsistency of yield simulation by APSIM for the different varieties used in this study, we were not able to test these disease files.

5.7. DISCUSSION

Application of crop models to evaluate plant growth requires sufficient details of cultivar type, soil, water, fertiliser and management regimes. In this study the performance of APSIM wheat model to simulate phenology, leaf area index, biomass and grain yield of winter wheat was evaluated under UK

conditions for two different field measurements data of Oakley and Cashel varieties. Our results showed that APSIM was unable to simulate the date of growth stages accurately in both varieties. The differences between the observed and simulated phenology was around 5-17 days. Phenology is a function of development rate that is controlled by temperature and photoperiod (Ritchie, 1991). In APSIM there are 11 phenological stages and accumulated thermal time is used to determine the duration of each stage (Robertson et al., 2002). In fact, accurate prediction of phenological development leads to accurate model simulation and determining crop yield is dependent on the length of the growing season and flowering time in relation to any stress (Stapper & Fischer, 1990). There are no UK winter wheat varieties that have been parameterised in APSIM and in this study we used a long season single winter wheat, cv. Claire, in the APSIM simulations. This may explain the differences in phenology stages between observed and simulations in this study, which may be due to the differences in the observed and used variety in the simulation.

All developmental stages were not simulated accurately with APSIM in this study in particular GS31. The occurrence of floral initiation and terminal spikelet's is related directly to the leaf appearance due to the fact that apical meristem development and leaf appearance are coordinated. Also leaf appearance rate and final number of leaves on the stem determines the flowering time (Jamieson et al., 1995). Therefore accurate phenology simulation is related to the accuracy in simulating the leaf initiation and leaf appearance. Bassu et al. (2009) found that using measured phyllochrons from

durum wheat grown in variable Mediterranean environment instead of a general value, improved the performance of APSIM-wheat model to simulate anthesis date and grain yield. The availability of the routine that is sensitive to the low temperature conditions of the UK is very important and this could be a further study to improve APSIM simulations. In addition, parameterisation of UK winter wheat in APSIM is needed to enhance the performance of the simulation.

Moreover, predications of crop biomass and leaf area index were overestimated by APSIM in both experimental years. In contrast yield was reasonably estimated in 2012/13, while under estimated in 2014/15, the predication was close to the yield of the diseased crop of the observed variety in this year. This result agreed with implementation of APSIM in temperate region in the Netherlands. APSIM simulated LAI poorly and yield was overestimated in particular with season of lower observed yield, while biomass simulation performed well (Asseng et al., 2000). Moreover, APSIM have been evaluated for the crop growth of phenology, LAI, biomass and yield of winter wheat in North China Plain (NCP). The model simulation was reasonably good to capture biomass, however relatively poor in simulation of LAI and could not accurately capture the grain or yield responses to different planting densities and sowing dates (Zhang et al., 2012).

Generally, prediction of the leaf area is more difficult than biomass. This problem seems to be common with different models not only APSIM. LAI of wheat crop was also overestimated when APSIM performance was evaluated

against wider range of field measurements in Western Australia (Asseng et al., 1998). Overestimation of wheat LAI evaluated in temperate regions was noted with APSIM in North West Europe (Asseng et al., 2000). On the other hand, other models like ORYZA2000 overestimated the LAI of rice grown under different range of N fertilizer condition in the Philippines (Bouman & van Laar, 2006). Moreover, a lower estimation of maize LAI was predicted by CERES-Maize model in a semi-arid Mediterranean environment (Ben Nouna et al., 2000). Winter wheat generally sown in the UK between September and November emerges before winter and enters in vernalisation over winter, then starts to grow again in February/March. Some leaves may die under extreme temperature during long winter period (Zhang et al., 2012). The lack of sufficient data about the response of green leaf death rate due to decreasing minimum temperature in winter wheat may was the cause to overestimation of LAI by APSIM. Further study is needed to quantify the leaf senescence and re-growth dynamics of winter wheat.

Agreement between field observation of biomass and APSIM simulation was relatively good with overestimation in both years. The same result has been achieved for APSIM simulation of wheat biomass in the Netherland and Western Australia (Asseng et al., 1998; Asseng et al., 2000). Crop model like APSIM use crop-specific radiation use efficiency (RUE) to estimate daily biomass production and allocating biomass to different organs using stage dependent empirical coefficients (Hammer & Muchow, 1994; Sinclair & Muchow, 1999). The equation to calculate biomass in APSIM as follows:

$$Biomass = 1 - \exp^{-KL} \times RUE,$$

where; $1 - \exp^{-KL}$ = light interception and

RUE= radiation use efficiency and the specific RUE value of wheat is 1.24 (Holzworth et al., 2014). LAI is critical for light interception and photosynthesis in the crop model (Ritchie et al., 1985). Therefore, the overestimation of biomass in this study is largely attributed to the poor prediction of leaf area and leaf area index.

Yield was under predicted by APSIM comparing to the healthy field observation in particular in the 2014/15. However, close simulation prediction was observed with diseased data, despite the fact that APSIM assumes no competition from weeds, pests or diseases. This result agrees with testing the Nwheat and Iwheat of APSIM model that revealed APSIM was not suitable for estimating wheat yields and proteins in southwest Queensland (Robinson et al., 2001). The limit of soil data available for APSIM parameterisation, in particular the absence of directly measured ‘crop lower limits’ of water extraction, and the absence of measured soil characteristics below 90cm may was the reason for the lower estimation of the yield. Therefore, more details about UK soil characteristics is highly needed to facilitate crop simulation modelling under UK environment.

In APSIM grain number per plant is estimated using stem dry matter at anthesis multiplied by a cultivar parameter “grains-per-gram-stem” with default value 25-grain g⁻¹. Grain weight increase is simulated with a potential grain growth rate as limited by biomass and translocation from start to end of grain filling. Grain number and final grain weight determines the yield (Holzworth et al., 2014). Moreover, a linear relation was found between grain

per plant and biomass growth during spikelet development stage (the stage after jointing and before flowering) (Duggan et al., 2000; Brancourt-Hulmel et al., 2003). Inaccurate simulation of phenology and leaf area index may explain the lower estimate of yield in this study. Therefore accurate simulation of phenology and biomass is needed to predict the yield accurately.

Generally, APSIM had poor prediction of the phenology, LAI, biomass and yield of winter wheat grown under UK conditions. This result led to the conclusion that the current version of APSIM model is not efficient to simulate wheat growth and development for temperate conditions of UK. Therefore, enhancing APSIM parameterisation and adding cultivar coefficients specific for UK wheat varieties are very important. Also understanding the APSIM simulation and required data and correct measurement from the experimental field is essential to obtain accurate predication from crop simulation.

Farming simulation models that are widely used today have progressed from specific crop model or soil model to incorporated soil and crop model (Moore et al., 2014). The lack of farming system models utilising biotic constraints sub-model is most likely due to the complexity and difficulty of parameterization and required knowledge of the models (Colbach, 2010). However, development of communications infrastructure allows APSIM to communicate with different software languages, simplifying the linking process with other models (Holzworth et al., 2010). While, APSIM simulation for UK wheat growth was not promising in this study.

The Common Modelling Protocol (CMP) in APSIM allows other models to integrate and simulate process within the farming system models using different language (Moore et al., 2007). This study used eyespot disease index data taken from disease inoculated trials observation between 2004 and 2014. The eyespot disease models implemented with APSIM simulated an adequate level of disease predication at GS13, GS32 and GS39. The IPM model was able to predict initial infection at GS13 with about 9.6% disease index. The DDM model also was able to predict generally the development of disease index at GS32 at about 1.3% and DSM at GS39 with about 12%. The r^2 deviance for IPM, DDM and DSM models was between 0.93-0.99. There have been few attempts to link disease models with crop simulation models with limited success in quantifying the disease effects on targeted crops. For example DYMEX disease was successfully linked with APSIM, however the linked models had limited ability to predict the proportions of disease in all examined years (Whish et al., 2015).

Harvest reduction model could not be looped with APSIM due to the un-settable variables of biomass and yield reduction in APSIM. Further development of APSIM to allow such function to be modified by model user is highly needed. Unfortunately, less availability of sufficient data prevented from testing extra models added to the workflow. Moreover, we were unable to use a co-efficient in the reduction files developed by Neil I. Huth, due to the yield not being simulated consistently for different varieties by APSIM in this study.

In fact estimation of specific value of the disease level is difficult due to the instability of the disease development and its dependence on environmental conditions and the crop host. However, this result demonstrated successful implementation of disease model with crop simulation model. Deep understanding of disease and crop interactions and the mechanics on how disease cause damage to the crop is very important, to allow future link between crop and disease model and better simulation of crop growth development and yield in response to the disease.

Chapter 6

6. GENERAL DISCUSSION

6.1. GENERAL DISCUSSION

Demand for food will increase with any further increase in population and change in consumption pattern. In many developing countries, crop yield is low therefore further improvement is necessary to meet future demands. Climate is an important component in the food production system and plays a key role in crop development and yield formation. Food production depends on local climatic conditions such as temperature and rainfall and any change in current conditions will result in altered productivity. Thus, producing food more efficiently and increasing wheat yields remain high priorities worldwide. In the United Kingdom for instance food production will be highly affected by unstable weather condition. According to the report (United Kingdom climate impacts program, 2011), summers are expected to be warmer in all areas across the UK with predicted temperature increases of 1.5 to 2°C. Moreover, change in precipitation patterns is expected to cause drier summers and wetter winters with predictions that rainfall will increase by 10% to 20% during winter.

Many pests and diseases are capable of relatively rapid genetic changes. Climate change may enhance their ability to invade new areas as well as alter their seasonal patterns and abundance (Clements & Ditommaso, 2011). Accordingly, survival, development, reproduction and dispersal of plant pathogens are dependent on climate to a certain degree. For example, survival of some pathogens is increased by mild winters and humid weather. Climate conditions can mediate changes to pest and disease populations that pose an enormous risk to crop yields and global food security. Change in climate can

cause changes in pathogen complexes impacting on crop yield, safety and quality. Fusarium head blight (FHB) disease in wheat is a good example of these effects (Chakraborty & Newton, 2011). Due to climate change, FHB re-emerged in the northern Great Plains and central USA between 1998 and 2000 causing yield loss and grain price reduction estimated by \$2.7 as a result of reducing grain quality (Goswami & Kistler, 2004). Beside FHB infection, grain loss can be also due to production of trichothecene mycotoxins and oestrogenic zearalenone in infected host tissue that is harmful to humans and animals.

Moreover, disease development as well as physiology and resistance of plant hosts can be altered by climate change. Plant canopy size and density can be increased significantly from higher levels of CO₂ that result in a high nutritional quality and a greater biomass (Manning & Tiedmann, 1995; Islam et al., 2012). However, these promote foliar diseases such as rusts, powdery mildew, leaf spots and blights particularly in the situation when excessive humidity exists in the canopy (Coakley et al., 1999; Islam et al., 2012). Despite the recent advancements in breeding and improvements in integrated pest management, crop losses due to pest and diseases are still high and can reach over 50% in the major crops and can be even higher under favourable conditions such as high temperature and high rainfall (Oerke, 2006). By way of illustration, Oerke (2006) analysed losses of 6 major crops between 2001 and 2003 and found that the average loss in wheat and cotton due to plant diseases was 29%, while in potato the loss was 40%.

The variation in loss from place to place and season to season is particularly due to variation in climate condition that influences the incidence and severity of the disease and pest (Flood, 2010). The accurate yield loss data caused by diseases on farmer's fields in the developing countries such as Oman are often absent or difficult to obtain. However, this is not the case in developed countries, for example in the UK where accurate information about disease and estimated losses is available. Modelling is an important tool to increase our understanding about the future impact of biotic and abiotic factors on crop production. A considerable amount of literature has been published on modelling approaches; however, few of these models include crop loss components due to disease. Such a component needs to be incorporated in the modelling to arrive at a realistic estimate of crop loss because of disease under climate change. Therefore, it is important to develop crop models predicting the reduction in crop production when attacked by diseases.

The overall aim of this project was to model disease impact on wheat for improved food security. The first objective was to compare the incidence of wheat diseases between 2009 and 2014 in two different agro-ecological zones, UK and Oman (chapter 2). Also, to identify the main disease threats and quantify their impact on wheat production in Oman using data of diseases in Omani wheat collected in 2014 survey. The occurrence and the incidences of fungal wheat diseases in two different climatic condition Oman and UK were compared between 2009 and 2014. In Oman, 447 fields in five different locations were assessed for stem and foliar disease incidence between 2009 and 2014 at GS 55-69, while in the UK almost 300 crops were assessed annually

for leaf, stem and ear diseases at the early milk development stage (GS73-75) between 2009-2014.

There was a variation in disease incidence of leaf spot, stem base diseases, loose smut and powdery mildew in Omani wheat at GS55-69 between 2009 and 2014. The predominant disease was leaf spot recorded at all growth stages during the 2014 survey as well as recorded with high frequency among other diseases and increasing through years. Stem base was the important disease of stems and loose smut was important disease of ears. In UK winter wheat the most widespread leaf disease between 2009 and 2014 was *Septoria*. The level of powdery mildew in UK wheat was more common and at a higher level than that recorded in Omani wheat at the same period. Fusarium was the most common disease of stem in UK wheat followed by eyespot. The lower level of eyespot recorded during the 6 years period of the survey may be related to the fungicide treatments applied at GS31 or difficulty to assess the lesion at GS73-75. During the 6 years survey in Oman eyespot was not found and this may be related to the high temperature during winter in Oman. Loose smut was the most common ear disease in Omani wheat while ear blight was the most important disease of the ears in UK winter wheat. Introduction of new irrigation systems may favour some disease like stem base in Oman that was recorded in high frequency in the last year of the survey.

The pathogens in Omani wheat were characterised and the influence of agronomy factors were assessed in five provinces (Buraimai, Thahira, Interior, Sharqia and Batinah). Isolations from six symptomatic wheat varieties in 2014 resulted in 36 different fungal species. The four most frequently isolated pathogens from infected tissues were *Alternaria alternata*, *Bipolaris sorokiniana*, *Setosphaeria rostrata*, and *Fusarium equiseti*. These fungi have been also been isolated from seeds of Omani wheat and only *B. sorokiniana* was found to cause root and crown rot in wheat (Al-Sadi & Deadman, 2010).

In this study *Setosphaeria rostrata* was recovered with high frequency from all growth stages and all locations covered by this survey. This result was supported by a survey that found *S. rostrata* causing blights, spots and blotches in wheat leaves in different growing area in India (Singh et al., 2001). *F. equiseti* was the only *Fusarium* species recovered from the ear samples from this study. *Fusarium* species isolated in this study have been reported as able to cause disease on stem bases, roots, leaves and ears (Liggitt et al., 1997; Narkiewicz-Jodko et al., 2003). Pathogenicity tests of *A. alternata*, *B. sorokiniana*, *S. rostrata* and *F. equiseti* revealed with no significant differences in disease caused on two wheat cultivars.

During 2014 survey agronomic practices on disease incidence on Omani wheat was also considered. It was found that stem base disease was influenced significantly by urea application. Leaf spot incidence was found to be lower in fields fertilized with urea application 2 months from sowing comparing to one month from sowing. Whereas, foliar application with potassium and

ammonium was influenced the incidence of loose smut diseases. This is supported by a study that found urea application to tomato plants influenced wilt caused by *F. oxysporum* f. sp. *lycopersici* (James, 1996). Buramiai province had the highest incidence of leaf spot disease while, Thahira province had higher incidence of stem-base diseases. However, the lowest incidence of leaf spot was recorded from Sharqia, whilst Batinah had the lowest incidence of stem base disease. It has been found from this study that some agronomic practices influenced disease incidence significantly at GS55-69. Leaf spot was found to be highest with mechanical sowing method, location (Sharqia province) and variety (W.Q.302). However, years, mechanical sowing method and drip irrigation had highest significant influences on stem base diseases. These results indicate that some practices influenced diseases of wheat in Oman like time and quantity of fertiliser. Also, the method of sowing need to be considered as mechanical always influenced the disease occurrence. Overall the identification of main disease threats in Oman and its agronomic influencer is the basis for further research. To determine the priority in disease problems, to assess the economic importance and to contrast environment model for yield loss caused by disease.

The effect of treatment on risk aversion to eyespot disease and cost recovery of eyespot treatment through yield response of the crop was assessed in chapter 3 of this study. The study confirmed that all treatments apart from epixiconazole (Opus at 1 l ha⁻¹) reduced eyespot disease at GS39. This study found that only boscalid and epoxiconazole (Tracker) fungicide gave a clear dose response resulting in greater yield and higher gross margin.

In chapter 4, epoxiconazole (Opus at 0.5 l ha^{-1}) returned better gross margin and yield compared to prothioconazole (Proline 275 at 0.4 l ha^{-1}) treatment. Most likely because epoxiconazole is very effective against other diseases such as *Septoria* and also it is much cheaper than prothioconazole. The yield and gross margin increased when fungicide treatments were applied compared to the control under both high and low disease pressure. The results in chapter 3 revealed that the treated trial means of yield and gross margin dominated the untreated trial means. In addition, in all years, except 2005 and 2006, treated yield and gross margin had lower range of standard deviation (± 2). This indicates that treating the disease is a better choice for the grower; besides it reduces the risk of high yield loss due to extreme disease severity. The dominant strategy for growers was to apply fungicides. Thus, although the mean gross margin of the untreated trials was more competitive, treatment would still be a better choice for grower, because there is less uncertainty about the outcome and less deviation. Furthermore, fungicide treatment of eyespot was found cost effective under high and low disease pressure situations.

The third objective was to develop an eyespot disease model predicting yield loss of wheat in the UK. This study found a significant positive relationship between thermal time, average relative humidity and disease infection (Chapter 4). While increased thermal time caused disease development and severity to decrease slightly, relative humidity caused disease development and severity to increase significantly. This finding implies that thermal time has different effects on the stages of diseases, possibly related on the crop itself. Since increased thermal time also results in increased crop development, which is

less favourable for disease development and severity, as some inoculum may be lost due to increased development through loss of infected leaf sheaths. This finding implies that development of monocyclic disease like eyespot can be measured by thermal time (Lovell et al., 2004).

The role of rain seems to be different than that with thermal time. Total rainfall from sowing to GS12/13 has negative effect on infection stage of disease, while total rainfall between GS31/32 to GS39 had a positive effect on penetration and establishment. This result is in agreement with previous work that showed eyespot incidence is influenced by high rainfall between March and May (Burnett & Hughes, 2004). Region had a major impact on eyespot, with the largest difference exerted in the West and North. Furthermore, disease infection decreased significantly in heavy soils but increased in light soils.

Additionally, the effect of previous crop had the largest positive influencing on disease index in all models. Non-host such as legumes reduced disease index significantly in IPM and DDM. This finding support previous work that showed eyespot was more severe if wheat occurred in a rotation with a high incidence of other cereals compared to rotations where non-cereal crops were regularly grown (Cook et al., 1991). Minimal tillage caused higher infection potential and disease development but not severity at GS39 where minimal tillage reduced disease in comparison to ploughing. The effect of minimal tilling at infection and development stages may be due to the presence of higher inoculum on debris left in minimal cultivation. However, at establishment stage it could be under min-till there may be other competitors,

which may alter disease severity. Late sowing dates after the 6th October also reduced disease at IPM and DDM. All fungicides applied at GS31/32 were also found to reduce disease significantly at GS39 in comparison to epoxiconazole alone. These results agreed with previous studies on fungicide effectiveness against eyespot disease (Cook, 1980; Ray et al., 2004). Overall cyprodinil (Unix) treatments had the lowest reduction in disease severity, whilst mixture of epoxiconazole and Boscalid had highest reduction in disease severity. This study found a significant relationship between disease severities at GS39 and yields loss. Under different scenario, yield was affected with disease incidence increase or decrease. For instance in a scenario were disease severity increase by 26%, yield reduced by 0.6 t/ha^{-1} .

The ability of APSIM to simulate the crop growth of two different winter wheat varieties (Oakley and Cashel) under UK conditions was evaluated. The development of both varieties was not accurately simulated by APSIM. The simulation revealed a significant gap between observed and simulated data, particularly at GS31 with a range between 5-17 days in all growth stages. Moreover, in both experimental years APSIM predictions of leaf area index were overestimated. In contrast, yield was reasonably estimated in 2012/13, while under estimated in 2014/15. APSIM simulation of wheat biomass in the Netherlands and Western Australia (Asseng et al., 1998; Asseng et al., 2000) has also been shown to be inaccurate. The general finding from the work in chapter 3 was that the inaccurate simulation of phenology and leaf area index might explain the lower estimate of biomass and yield. The poor prediction of the phenology, LAI, biomass and yield of winter wheat grown under UK

conditions APSIM had in this study led to the conclusion that the current version of the APSIM model is not efficient to simulate wheat growth and development for temperate conditions of the UK.

However, eyespot disease models developed in chapter 4 were implemented with APSIM and simulated an adequate level of disease prediction at GS13, GS32 and GS39. The literature shows that few attempts have been made to link APSIM with disease models. APSIM was successfully linked with DYMEX disease model but had limited ability to predict the proportions of disease in all examined years (Whish et al., 2015). In this work, yield loss could not be quantified due to un-settable variables of biomass and yield reduction in APSIM prevented from looped harvest reduction model. Thus the developed models in the workflow in chapter 4 could not be tested due to insufficient data and yield not being simulated consistently for different varieties by APSIM.

6.2 Future studies

- The main diseases threatening wheat production in Oman identified in chapter 2 of this study was the first step towards using crop modelling approach to aid in enhancing wheat production and quantifying yield loss due to diseases. Future study is highly needed to determine the priority in disease problems, plan for future research to assess the economic importance and to contrast environment model for yield loss caused by disease as well as developing effective integrated disease management strategies for Omani wheat.

- In chapter 4 of this study a significant correlation between yield loss and eyespot severity at GS39 has been found that is contrasted with no consistent correlation between yield loss and eyespot severity result found by Burnett et al. (2012). This indicated the need of further research to investigate the role of eyespot disease on yield.
- Reduction of disease under scenario of increasing environmental factors found in chapter 4 of this study implies that under future climate change and increase in parameters like temperature and rainfall might decrease the distribution and severity of eyespot. Further work is needed to understand the relationship between environmental factors under and eyespot disease development climate change.
- Cultivar was not considered within the factor that effect disease development models. Further research to include a range of variety resistance rating and check their effect upon eyespot disease.
- Fertiliser application was not considered. Evidence from literature considering this factor was contrasting. While Colbach and Saur (1998) found significant less eyespot in plots fertilised with ammonium sulphate than those fertilised with ammonium nitrate. However, Smith et al. (2000) found no response from eyespot with addition of ammonium nitrate at different doses. Therefore, fertiliser factor may be included in the model as future study to investigate its effect upon eyespot disease and yield.
- The inaccurate prediction of winter wheat crop growth simulation by APSIM under UK condition in chapter 5 of this study implies the need of further work in model structure and measurement of field data.

- In the model side, availability of the routine that is sensitive to the low temperature conditions of the UK is very important and this could be a further study to improve APSIM simulations.
- Enhancing APSIM parameterisation and adding cultivar coefficients specific for UK wheat varieties of are very important.
- The lack of sufficient data about the response of green leaf death rate due to decreasing minimum temperature in winter wheat may was the cause to overestimation of LAI by APSIM. Further study is needed to quantify the leaf senescence and re-growth dynamics of winter wheat.
- In the field measurement, the limit of soil data available for APSIM parameterisation and the absence of measured soil characteristics below 90 cm was one of the reasons that caused inconsistency of yield simulation. More work is needed about UK soil characteristics to facilitate crop simulation modelling under UK environment.
- Understanding APSIM simulation and correct measurement from the experimental field is essential to obtain accurate predication from crop simulation. Future work to understand the disease and crop interactions and the mechanics on how disease cause damage to the crop is very important, to allow future link between crop and disease model and to enhance simulation of crop growth development and yield in response to the disease.

REFERENCES

- AINSWORTH, E., A. & LONG, S., P. 2005. What have we learned from 15 years of free-air CO₂ enrichment (FACE)? A meta-analytic review of the responses of photosynthesis, canopy properties and plant production to rising CO₂. *New Phytologist* 165, 351–72.
- AL-AZRI, M., S. LEIBOVICI, D. KARUNARATNE, A. MEEK, S. & RAY, R., V. 2014. Simulating eyespot disease development and yield loss using APSIM for UK wheat. Agriculture and climate change. Adapting crops to increased uncertainty, Amsterdam, The Netherlands, 15-17 February 2015.
- AL-SA'DI, A., M. DRENTH, A. DEADMAN M. DE COCK, A., W., A., M. & AITKEN, E., A., B. 2007. Molecular characterization and pathogenicity of *Pythium* species associated with damping-off in greenhouse cucumber (*Cucumis sativus* L.) in Oman. *Plant Pathology* 56:140-149.
- AL-SADI, A., M. & DEADMAN, M., L. 2010. Influence of Seed-borne *Cochliobolus sativus* (*Anamorph Bipolaris sorokiniana*) on Crown Rot and Root Rot of Barley and Wheat. *Journal of Phytopathology* 158 (10): 683-690.
- AL-SADI, A., M. AL-MASOODI, R., S. AL-ISMAILI, M. & AL-MAHMOOLI, I., H. 2015. Population structure and development of resistance to hymexazol among *Fusarium solani* populations from date palm, Citrus and cucumber. *Journal of Phytopathology*. 163:947–955.
- ALBERTINI, C. GREDT, M. & LEROUX, P. 2003. Polymorphism of 14 alpha-demethylase gene (CYP51) in the cereal eyespot fungi *Tapesia acuformis* and *Tapesia yallundae*. *European Journal of Plant Pathology* 109:117-128
- ALCAMO, J., D. VAN VUUREN. C. RINGLER. W. CRAMER, T. MASUI, J., A. & SCHULZE, K. 2005. Changes in nature's balance sheet: model-based estimates of future worldwide ecosystem services. *Ecology and Society*, 10(2), 19.
- AL-LAWATI, A., H. & NADAF, S., K. 2001. Focus on Seed Programs. The Seed Industry in Oman. A series of country reports published by the WANA Seed Network Secretariat, Seed Unit, ICARDA.
- ANONYMOUS. 2010. The Future of World Food Security. Rome, Italy: International Fund for Agricultural Development.

- ANTLE, J., M. 1983. Incorporating Risk in Production Analysis. *American Journal of Agricultural Economics*, 65, 1099-1106.
- ARSHAD, M., A. GILL, K., S. & COY, G., R. 1994. Wheat Yield and Weed Population as Influenced by 3 Tillage Systems on a Clay Soil in Temperate Continental Climate. *Soil & Tillage Research*, **28**, 227-238.
- ASSENG, S. FOSTER I. & TURNER, N., C. 2011. The impact of temperature variability on wheat yields. *Global Change Biology* 17, 997–1012.
- ASSENG, S. KEATING, B., A. FILLERY, I., R., P. GREGORY, P., J. BOWDEN, J., W. TURNER, N., C. PALTA, J., A. & ABRECHT, D., G. 1998. Performance of the APSIM-wheat model in Western Australia. *Field Crops Res.* 57: 163-179.
- ASSENG, S. VAN KEULEN, H. & STOL, W. 2000. Performance and application of the APSIM N-wheat model in the Netherlands. *European Journal of Agronomy*, 12, 37-54.
- BASSO, B., J., T. RITCHIE. F., J. PIERCE. R., P. BRAGA, & JONES, J., W. 2001. Spatial validation of crop models for precision agriculture. *Agriculture System*. 68: 97–112.
- BASSU, S. ASSENG, S. MOTZO, R. & GIUNTA, F. 2009. Optimising sowing date of durum wheat in a variable Mediterranean environment. *Field Crops Research*. 111: 109-118.
- BATEMAN, G., L. EDWARDS, S., G. MARSHALL, J. MORGAN, L., W. NICHOLSON, P. NUTTALL, M. PARRY, D., W. SCHRANCHER, M. & TURNER, A., S. 2000. Effects of cultivar and fungicides on stem-base pathogens, determined by quantitative PCR, and on diseases and yield of wheat. *Annals of Applied Biology*, 137, 213-221.
- BATEMAN, G., L. & TAYLOR, G., S. 1976. Significance of the coleoptile in establishment of seedling infection on wheat by *pseudocercospora herpotrichoides*. *Transactions of the British Mycological Society* 67: 513-514.
- BAUR, H., P. & SCHMITT, W. 2004. Prothioconazole – a new dimension DMI Biochemistry, mode of action, systemic effects. *Pflanzenschutz-Nachrichten Bayer*, 57, 237-248.
- BEN NOUNA, B. KATERJI, N. & MASTRORILLI, M. 2000. Using the CERES-Maize model in a semi-arid Mediterranean environment. Evaluation of model performance. *European Journal of Agronomy*. 13:309-322.

- BERNDES, G. 2002. Bioenergy and water—the implications of large-scale bioenergy production for water use and supply. *Global Environmental Change*, 12, 253–271.
- BLACK, T., A. GARDNER, W., R. & TANNER, C., B. 1970. Water Storage and Drainage under a Row Crop on a Sandy Soil. *Agronomy Journal*, 62, 48-55.
- BLEIN, M. LEVREL, A. LEMOINE, J. GAUTIER, V. CHEVALIER, M. & BARLOY, D. 2009. *Oculimacula yallundae* lifestyle revisited: relationships between the timing of eyespot symptom appearance, the development of the pathogen and the responses of infected partially resistant wheat plants. *Plant Pathology*, 58, 1-11.
- BOCK, A., M. WAN, A., M. & FITT, B., D., L. 2009. Development of *Oculimacula yallundae* and *O. acufomis* (eyespot) lesions on stems of winter wheat in relation to thermal time in the UK. *Journal of Plant Pathology*, 58: 12-22.
- BOOTE, K., J. & JONES, J., W. 1998. Simulation of crop growth: CROPGRO model. *Agricultural Systems Modelling and Simulation*, R. M. Peart and R. B. Curry., Eds., Marcel Dekker, 651–692.
- BOOTE, K., J. JONES, J., W. MISHOE, J., W. & BERGER, R., D. 1983. Coupling pests to crop growth simulators to predict yield reduction. *The American Phytopathological Society*, 73. 11: 1581-1587.
- BOTTALICO, A. & PERRONE, G. 2002. Toxigenic *Fusarium* species and mycotoxins associated with head blight in small-grain cereals in Europe. *European. Journal of Plant Pathology*. 108:611-624.
- BOUMAN, B., A., M. & VAN LAAR, H., H. 2006. Description and evaluation of the rice growth model ORYZA2000 under nitrogen-limited conditions. *Agriculture System*. 87: 249-273.
- BRANCOURT-HULMEL, M. DOUSSINAULT, G. LECOMTE, C. BE'ARD, P., L., E. BUANEC, B. & TROTTET, M. 2003. Genetic improvement of agronomic traits of winter wheat cultivars released in France from 1946 to 1992. *Crop Science*. 43: 37-45.
- BRINKMAN, H. & CULLEN, S. & HENDRIX, S. 2011. Food Insecurity and Violent Conflict: Causes, Consequences, and Addressing the Challenges. Occasional Paper n° 24. World food programme. Wfp.org.

- BROWN, M. 2009. Markets, climate change, and food security in West Africa. *Environment Science and Technology*, 43 (2): 8016-8020.
- BROWNLIE, J., C. PECKHAM, J. WAAGE, M. WOOLHOUSE, C. LYALL, L. MEAGHER. J. TAIT, M. BAYLIS. & NICOLL, A. 2006. *Foresight. Infectious Diseases: preparing for the future Future Threats*. Office of Science and Innovation, London.
- BURNETT, F., J. & OXLEY, S., J., P. 1996. The importance and control of common eyespot in wheat *Proceedings Crop Protection in Northern Britain*, 1996, 1, 121 - 126.
- BURNETT, F., J. 1999. The use of fungicide sequences to maximise the control of eyespot in cereals and minimise the risk of sharp eyespot. Project Report No. 200. Home Grown Cereals Authority, London.
- BURNETT, F., J. & HUGHES, G. 2004. The development of a risk assessment method to identify wheat crops at risk from eyespot. Project Report No. 347. Home Grown Cereals Authority, London.
- BURNETT, F., J. 2005. Validation of a risk assessment method to identify wheat crops at risk from eyespot. HGCA Project Report No. 347 Part 2. Home Grown Cereals Authority, London.
- BURNETT, F., J. OXELEY, S., J., P. & LAING, A., P. 2000. The use of PCR diagnostics in determining eyespot control strategy in Wheat. BCPC Conference: Pests & diseases 2000, 1-3, 107-112.
- BURNETT, F. BUTLER-ELLIS, C. GARETH HUGHES, G. KNIGHT, S. & RUMIANA, R. 2012. Forecasting eyespot development and yield losses in winter wheat. Project Report No. 491. Home Grown Cereals Authority, London.
- BUTTERWORTH, M., H., M., A. SEMENOV. A. BARNES, D. MORAN, J., S. WEST. & FITT, B., D., L. 2010. North-south divide; contrasting impacts of climate change on crop yields in Scotland and England. *Journal of the Royal Society Interface* 7:123-130.
- CADLE, M., M. MURRAY, T., D. & JONES, S., S. 1997. Identification of resistance to *Pseudocercospora herpotrichoides* in *Triticum monococcum*. *Plant Disease*, **81**, 1181-1186.
- CASSEY, H. 1973. Farm planning and control, *Journal of Agricultural Economics*, 24, 606-607.

- CLEMENTS, D., R. & DITOMMASO, A. 2011. Climate change and weed adaptation: can evolution of invasive plants lead to greater range expansion than forecasted? *Weed Research*, 51: 227–240.
- CHAKRABORTY, S., A., C. & NEWTON, A., S. 2011. Climate change, plant diseases and food security: an overview. *Plant Pathology*, 60: 2–14.
- CHAKRABORTY, S. TIEDEMANN, A., V. & TENG, P., S. 2000. Climate change: potential impact on plant diseases *Environmental Pollution*. 108: 317–326.
- CHALLINOR, A., J., J., M. SLINGO, T., R. WHEELER, P., Q. GRAUFURD, & GRIMES, D. I., F. 2003. Toward a Combined Seasonal Weather and Crop Productivity Forecasting System: Determination of the Working Spatial Scale. *Journal of Applied Meteorology*, 42: 175-192.
- CHALLINOR, A., J., T., R. WHEELER. P., Q. CRAUFURD. J., M. SLINGO. & GRIMES, D., I., F. 2004. Design and optimization of a large process- based model for annual crops. *Agricultural and Forest Meteorology*, 124: 99-120.
- CHEN, C. WANG, E., L., Y., U., Q. & ZHANG, Y., Q. 2010. Quantifying the effects of climate trends in the past 43 years (1961_2003) on crop growth and water demand in the North China Plain. *Climate Change*. 100: 559-578.
- CHEN, R., S. 1990. 'Global agriculture, environment, and hunger: past present and future links', *Environmental Zmpacl Assessment Review*, 10: 335-358.
- CHEN, R., S. & KATES, R., W. 1994. World food security: prospects and trends. *Food Policy*, 19 (2): 192-208.
- CHRISTENSEN, J., B. HEWITSON. & MEARNES, O. 2007. Regional Climate Projections. In *Climate Change 2007: The Physical Science Basis: Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change*; Solomon, S., Qin, D., Manning, M., Eds.; Cambridge University Press: Cambridge, UK.
- CLARKSON, J., D., S. 1981. Relationship between Eyespot Severity and Yield Loss in Winter-Wheat. *Plant Pathology*, 30, 125-131.
- CLARKSON, J., D., S. & POLLEY, R., W. 1981. Assessment of losses caused by stem base and root diseases in cereals. *Proceeding of the 1981 British crop protection conference- pests and diseases* 1:223-231.

- CLEMENTS, D., R. & DITOMMASO, A. 2011. Climate change and weed adaptation: can evolution of invasive plants lead to greater range expansion than forecasted? *Weed Research*, 51: 227–240.
- COAKLEY, S., M. SCHERM, H. & CHAKRABORTY, S. 1999. CLIMATE CHANGE AND PLANT DISEASE MANAGEMENT. *Annual Review of Phytopathology*. 37:399-426.
- COLBACH, N. & MEYNARD, J., M. 1995. Soil Tillage and Eyespot - Influence of Crop Residue Distribution on Disease Development and Infection Cycles. *European Journal of Plant Pathology*, 101, 601-611.
- COLBACH, N. & SAUR, L. 1998. Influence of crop management on eyespot development and infection cycles of winter wheat. *European Journal of Plant Pathology*, 104, 37-48.
- COLBACH, N. 2010. Modelling cropping system effects on crop pest dynamics: how to compromise between process analysis and decision aid. *Plant Science*. 179: 1-13.
- COLBACH, N. MEYNARD, J. M. DUBY, C. & HUET, P. 1999. A dynamic model of the influence of rotation and crop management on the disease development of eyespot. Proposal of cropping systems with low disease risk. *Crop Protection*, 18, 451-461.
- COOK, R., J. 1993. Eyespot - agronomic influences in the United Kingdom. In *exploring the depths of eyespot*. Ed. G D Palmer, Shering AG, Berlin, 83 - 89.
- COOK, R., J. 1980. Effects of late seasons fungicides spray on yield of winter wheat. *Plant pathology*. 29: 21-27.
- COOK, R., J. POLLEY, R., W. & THOMAS, M., R. 1991. Disease-Induced Losses in Winter-Wheat in England and Wales 1985-1989. *Crop Protection*. 10: 504-508..
- COOPER, P., J. DIMES, K. RAO. B., SHAPIRO. & TWOMLOW, S. 2008. Coping better with current climatic variability in the rain-fed farming systems of Sub-Saharan Africa: An essential first step in adapting to future climate change?. *Agriculture, Ecosystems & Environment*, 126 (1): .24-35.
- CRAWLEY, M., J. 2005. *Statistics: an introduction using R*, J. Wiley.
- CROOK, M., J. & ENNOS, A., R. 1995. The effect of nitrogen and growth regulators on stem and root characteristics associated with lodging in tow cultivars of winter wheat. *Journal of Experimental Botany* 46, 931-8.

- CROUS, P., W. EWALD GROENEWALD, J., Z. & GAMS, W. 2003. Eyespot of Cereals Revisited: ITS phylogeny Reveals New Species Relationships. *European Journal of Plant Pathology*, 109, 841-850.
- CURTIS, B., C. RAJARAM, S. & GOMEZ MACPHERSON, H. 2002. Bread wheat, improvement and production. Food and agriculture organization of the United Nations Rome.
- DANIELS, A. 1993. Early infection process of *Pseudocerosporella herpotrichoides* pathotypes. In exploring the depth of eyespot. Ed. G D palmer, Shering AG, Berlin. 29-37.
- DAVISON, A., C. 2003. *Statistical Models*, Cambridge University Press.
- DEADMAN, M., L. AL-SA_DI, A., M. AL-MAQBALI, Y., M. & AIME, M., C. 2007. First report of *Puccinia triticina* on wheat in Oman. *Plant Disease*. 91:113.
- DEBOER, R., F. STEED, G., R. KOLLMORGEN, J., F. & MACAULEY, B., J. 1993. Effects of Rotation, Stubble Retention and Cultivation on Take-All and Eyespot of Wheat in Northeastern Victoria, Australia. *Soil & Tillage Research*, 25 : 263-280.
- DEFRA. 2015. *Agricultural Land Use* [Online]. Available at: <http://www.defra.gov.uk/statistics/files/defra-stats-observatory-indicators-c2.pdf> [Accessed April 2nd 2016].
- Djumaniyazova, Y. Sommer, R. Ibragimov, N. Ruzimov, J. Lamers, J. & Vlek, P. 2010. Simulating water use and N response of winter wheat in the irrigated floodplains of Northwest Uzbekistan. *Field Crops Research* 116: 239-251.
- DUGGAN, B., L. DOMITRUK, D., R. & FOWLER, D., B. 2000. Yield component variation in winter wheat grown under drought stress. *Canadian Journal of Plant Science*. 80: 739-745.
- DUVEILLER, E. SINGH, R., P. & NICOL, J., M. 2007. The challenges of maintaining wheat productivity: pests, diseases, and potential epidemics. *Euphytica*. 157:417–430
- Evans, N. Baierl, A. Semenov, M., A. Gladders, P. Fitt, B., D., L. 2008. Range and severity of a plant disease increased by global warming. *Journal of the Royal Society Interface*. 5: 525-531.

- EVANS, N. BUTTERWORTH, M., H. BAIERL, A. SEMENOV, M., A. WEST, J., S. BARNES, A. MORAN, D. FITT, B., D., L. 2010. The impact of climate change on disease constraints on production of oilseed rape. *Food Security* 2:143-156.
- FAO, 2008. Food Outlook: Global Market Analysis. FAO, Rome.
- FAO, 2003. Trade Reforms and Food Security. Conceptualizing the Linkages. Rome, Italy: Food and Agriculture Organisation of the United Nations. <http://www.fao.org/docrep/005/y4671e/y4671e00.htm>.
- FAO. 2000. The state of food insecurity in the world (SOFI). Rome, Italy: FAO, UN. www.fao.org/FOCUS/E/SOFI00/sofi001-e.htm.
- FAOSTAT. 2016. Foods and Agriculture Organisation of the United Nations. <http://faostat.fao.org>
- FIRTH, C. 2002. The use of gross and net margins in the economic analysis of organic farms. Proceedings of the Organic Research 2002 Conference. 285-288.
- FISCHER, G. SHAH, M. & VAN VELTHUIZEN, H. 2002. Climate change and Agricultural vulnerability. Technical Report. International Institute for Applied Systems analysis. Available at <http://www.iiasa.ac.at/Research/LUC/>.
- FITT, B., D., L. BRUN, H. BARBETTI, M., J. & RIMMER, S., R. 2006. World-wide importance of phoma stem canker (*Leptosphaeria maculans* and *L. biglobosa*) on oilseed rape (*Brassica napus*) *European Journal of Plant Pathology*. 114. 3–15. doi:10.1007/s10658-005-2233-5.
- FITT, B., D., L. & BAINBRIDGE, A. 1983. Dispersal of *Pseudocercospora herpotrichoides* spores from infected wheat straw. *Journal of phytopathology* 106: 214-225.
- FITT, B., D., L. & NIJMAN, D., J. 1983. Quantitative studies on dispersal of *Pseudocercospora herpotrichoides* spores from infected wheat straw by simulated rain. *Netherlands Journal of plant pathology* 89: 198-202.
- FITT, B., D., L. & WHITE, R., P. 1988. Stages in the Progress of Eyespot Epidemics in Winter-Wheat Crops. *Zeitschrift Fur Pflanzenkrankheiten Und Pflanzenschutz-Journal of Plant Diseases and Protection*, 95: 35-45.

- FITT, B., D., L. GOULDS, A. & POLLEY, R., W. 1988. Eyespot *Pseudocercospora herpotrichoides* epidemiology in relation to prediction of disease severity and yield loss- a review. *Plant Pathology* 37: 311-328.
- FITT, B., D., L. GOULDS, A. HOLLINS, T., W. & JONES, D., R. 1990. Strategies for Control of Eyespot (*Pseudocercospora-Herpotrichoides*) in Uk Winter-Wheat and Winter Barley. *Annals of Applied Biology*, 117, 473-486.
- FLOOD, J. 2010. The importance of plant health to food security. *Food Security*. 2: 215–231. DOI 10.1007/s12571-010-0072-5.
- FREER, M. MOORE, A., D. & DONNELLY, J., R. 1997. GRAZPLAN: decision support systems for Australian grazing enterprises dII. The animal biology model for feed intake, production and reproduction and the GrazFeed DSS. *Agriculture System*.
- GARRETT, S., D. 1975. Cellulolytic rate and competitive saprophytic colonization of wheat straw by foot-rot fungi. *Soil Biology and Biochemistry* 7: 323-327.
- GAYDON, D., S. PROBERT, M. BURESH, R., J. MEINKE, H., SURIADI, A. DOBERMANN, A. BOUMAN, B. & TIMSINA, J. 2012. Rice in cropping systems- Modelling transitions between flooded and non-flooded soil environments. *European Journal of Agronomy*. 39: 9-24.
- GOSWAMI, R., S. & KISTLER, H., C. 2004. Heading for disaster: *Fusarium graminearum* on cereal crops. *Molecular Plant Pathology* 5: 515–25.
- GOULDS, A. & FITT, B., D., L. 1990. The development of eyespot on seedling leaf sheaths in winter wheat and winter barley crops inoculated with W-type and R-type isolates of *Pseudocercospora herpotrichoides*. *Journal of Phytopathology* 130, 161-173.
- GOULDS, A. & POLLEY, R., W. 1990. Assessment of eyespot and other stem base diseases of winter wheat and winter barley. *Mycological Research*, 94, 819-822.
- GOULDS, A. & FITT, B., D., L. 1991. Prediction of Eyespot Severity on Winter-Wheat or Winter Barley Inoculated with W-Type or R-Type Isolates of *Pseudocercospora-Herpotrichoides*. *Journal of Phytopathology*, 132, 105-115.
- GREGORY, P., J. JOHNSON, S., N. NEWTON, A., C. & INGRAM, J., S. 2009. Integrating pests and pathogens into the climate change/food security debate. *Journal of Experimental Botany*, 60 (10): 2827-38. doi: 10.1093/jxb/erp080.

- GREGORY, P., J. INGRAM, J., S., I. GOUDRIAAN, J. HUNT, T. LANDSBERG, J. LINDER, S. STAFFORD-SMITH, M. SUTHERST, R. & VALENTIN, C. 1999. Managed production systems. In: Walker, B. H., Steffen, W. L., Canadell, J. & Ingram, J. S. I. editors. The terrestrial biosphere and global change: implications for natural and managed systems. Cambridge: Cambridge University Press; 229-270.
- GUERIF, M. & DUKE, C., L. 2000. Adjustment procedures of a crop model to the site specific characteristics of soil and crop using remote sensing data assimilation. Agriculture Ecosystem Environment. 81 (1): 57–69.
- GUTTERIDGE, R., J. & HORNBY, D. 2003. Effects of sowing date and volunteers on the infectivity of soil infested with *Gaeumannomyces graminis var. tritici* and on take-all disease in successive crops of winter wheat. Annals of Applied Biology, 143, 275-282.
- HADAR, J. & RUSSELL, W., R. 1969. 'Rules for ordering uncertain prospects'. American Economic Review, 49:25-34.
- Hajihassani, M. Hajihassani, A. & Khaghani, S. 2012. Incidence and distribution of seed-borne fungi associated with wheat in Markazi Province, Iran. African Journal of Biotechnology. 11 (23): 6290-6295.
- HAMMER, G., L. & MUCHOW, R., C. 1994. Assessing climatic risk to sorghum production in water-limited subtropical environments. I. Development and testing of a simulation model. Field Crops Research 36: 221-234.
- HANOCH, G. & LEVY, H. 1969. 'Efficiency analysis of choices involving risk'. Review of Economic Studies. 36: 335-345.
- HARDAKER, J., B. 2006. "Farm risk management: past, present and prospects", Journal of Farm Management, 12 (10): 593-612.
- HARDAKER, J., B. HUIRNE, R., M., B. ANDERSON, J. R., & LIEN, G. 2004. Coping with Risk in Agriculture, 2nd edn, CAB International.
- HARDWICK, N., V. JONES, D., R. & SLOUGH, J., E. 2001. Factors affecting diseases of winter wheat in England and Wales, 1989–98. Plant Pathology, 50: 453-462.

- HARDWICK, N., V. JONES, D., R. & SLOUGH, J., E. 2001. Factors affecting diseases of winter wheat in England and Wales, 1989-98. *Plant Pathology*. 50: 453-462.
- HARDWICK, N., V. JONES, D., R. & SLOUGH, J., E. 2001. Factors affecting diseases of winter wheat in England and Wales, 1989-98. *Plant Pathology*, 50: 453-462.
- HARRELL, F., E. 2001. Regression modeling strategies: with applications to linear models, logistic regression and survival analysis, Springer.
- HENRY, R., S. JOHNSON, W., G. & WISE, K., A. 2011. The impact of fungicide and insecticide on soybean growth, yield and profitability. *Crop protection*, 30: 1629-1634.
- HGCA. 2012. Winter wheat HGCA Recommended List 2012/13 [Online]. Available at: http://www.hgca.com/document.aspx?fn=load&media_id=7369&publicationId=5880
- HIGGINS, S. & FITT, B., D., L. 1984.' Production and pathogenicity to wheat of *Pseudocercospora herpotrichoides* conidia. *Journal of Phytopathology*. 111: 222-231.
- HIGGINS, S., & FITT, B., D. L. 1985. 'Pathogenicity of *Pseudocercospora herpotrichoides* isolates to wheat seedlings and adult plants' *Zeitschrift fuer Pflanzenkrankheiten und Pflanzenschutz*. 92:176-185.
- HIJMANS, R., J. GUARINO, L. CRUZ, M. & ROJAS, E. 2001. Computer tools for spatial analysis of plant genetic resources data: 1. DiVA-GIS. *Plant Genetic Resources Newsletter*. 127: 15-19.
- HOCART, M., J. & MCNAUGHTON, J., E. 1994. Interspecific hybridisation between *Pseudocercospora herpotrichoides* and *P. anguioides* achieved through protoplast fusion. *Mycological Research*. 98: 47-56.
- HOFFERT, M., I. CALDEIRA, K. BENFORD, G. CRISWELL, D., R. GREEN, C. HERZOG, H. JAIN, A., K. KHESHGI, H., S. LACKNER, K., S. & LEWIS, J., S. 2002. Advanced technology paths to global climate stability: energy for a greenhouse planet. *Science*. 298:981-987.

- HOLLINS, T., W. & SCOTT, P., R. 1980. Epidemiology of eyespot (*Pseudocercospora herpotrichoides*) on winter wheat, with particular reference to the period of infection. *Annals of Applied Biology*. 95:19-29.
- HOLLOMON, D., W. 2012. Do we have the tools to manage resistance in the future? *Pest Management Science*, 68: 149–154.
- HOLZWORTH, D., P. HUTH, N., I. DEVOIL, P., G. ZURCHER, E., J. HERRMANN, N., I. MCLEAN, G. CHENU, K. VAN OOSTEROM, E., J. SNOW, V. MURPHY, C. MOORE, A., D. BROWN, H. WHISH, J., P., M. VERRALL, S. FAINGES, J. BELL, L., W. PEAKE, A., S. POULTON, P., L. HOCHMAN, Z. THORBURN, P., J. GAYDON, D., S. DALGLIESH, N., P. RODRIGUEZ, D. COX, H. CHAPMAN, S. DOHERTY, A. TEIXEIRA, E. SHARP, J. CICHOTA, R. VOGELER, I., LI, F., Y. WANG, E. HAMMER, G., L. ROBERTSON, M., J. DIMES, J., P. WHITBREAD, A., M. HUNT, J. VAN REES, H. MCCLELLAND, T. CARBERRY, P., S. HARGREAVES, J., N., G. MACLEOD, N. MCDONALD, C. HARSDORF, J. WEDGWOOD, S. & KEATING, B., A. 2014. APSIM - Evolution towards a new generation of agricultural systems simulation. *Environmental Modelling & Software*. 62, 327–350.
- HOLZWORTH, D., P. HUTH, N., I. & DE VOIL, P., G. 2010. Simplifying environmental model reuse. *Environmental Modelling & Software*. 25:269-275
- HOUGHTON, J., T., Y. DING, D., J. GRIGGS, M. NOGUER, P., J. LINDEN, & XIAOSU, D. 2001. The scientific basis, in Contribution of Working Group to the Third Assessment Report of the Intergovernmental Panel on Climatic Change (IPCC), p. 944, Cambridge Univ. Press.
- HUGHES, G. MCROBERTS, N. & BURNETT, F., J. 1999. Decision making and diagnosis in disease management. *Plant Pathology*, 48, 147-153.
- INGRAM, J., S., I. GREGORY, P., J. & IZAC, A., M. 2008. The role of agronomic research in climate change and food security policy. *Agriculture, Ecosystems and Environment*. 126 : 4–12.
- ISLAM, M., T. HOSSAIN, M., M. CLARKE, M., L. & AKANDA, M., A., M. 2012. Adaptation to Climate Change: Biodiversity, Food Security, Environmental Management and Rural Resilience in Bangladesh. In: ZAHID, A, QUMRUL HASSAN, M., RAHMAN, A., SALIM KHAN, M., ABDUL HASHEM, M. and

- LUTFUL HASSAN, eds., Impact of Climate Change on Water Resources and Food Security in Bangladesh AAGUB, Dhaka. 79-101
- JALALUDDIN, M. & JENKYN, J., F. 1996. Effects of wheat crop debris on the sporulation and survival of *Pseudocercospora herpotrichoides*. Plant Pathology. 45:1052-1064.
- JAMES, W., C. 1971. An Illustrated series of assessment keys for plant diseases, their preparation and usage. CAN. Plant Diseases. 51: 2.
- JAMES, R., L. 1996. Effects of Fertilizer on Selected Potential Plant Pathogens in Bareroot Forest Nurseries. USDA Forest Service, Forest Health Protection Coeur d'Alene, ID.
- JAMES, C. 1998. Global food security. Abstract. International. Congress. Plant Pathology. 7th Edinburgh, UK, Aug. No. 4.1GF. <http://www.bspp.org.uk/icpp98/4/1GF.html/>.
- JAMIESON, P., D. BROOKING, I., R. PORTER, J., R. & WILSON, D., R. 1995. Prediction of leaf appearance in wheat: a question of temperature. Field and Crops Research. 41: 35-44.
- JAMIESON, P., D. SEMENOV, M., A. BROOKING, I., R. & FRANCIS, G., S. 1998. Sirius: a mechanistic model of wheat response to environmental variation. European Journal of Agronomy, 8, 161–179.
- JAMIESON, P., D. PORTER, J., R. GOUDRIAAN, J. RITCHIE, J., T. KEULEN, H. & STOL, W. 1998. A comparison of the models AFRCWHEAT2, CERES-Wheat, Sirius, SUCROS2 and SWHEAT with measurements from wheat grown under drought. Field and Crop Research, 55: 23-44.
- JENKYN, J., F. GUTTERIDGE, R., J. BATEMAN, G., L. & JALALUDDIN, M. 2010. Effects of crop debris and cultivations on the development of eyespot of wheat caused by *Oculimacula* spp. Annals of Applied Biology, 156, 387-399.
- JONES, D., R. 1994. Evaluation of Fungicides for Control of Eyespot Disease and Yield Loss Nationships in Winter-Wheat. Plant Pathology, 43, 831-846.
- JONES, D., R. 1995. Timing of Fungicide Application for Control of Eyespot Disease of Winter-Wheat. Crop Protection, 14, 247-256.

- JONES, D. & BARNES, E., M. 2000. Fuzzy composite programming to combine remote sensing and crop models for decision support in precision crop management. *Agriculture System*. 65 (3): 137–158.
- JONES, P. & THORNTON, P. 2009. Croppers to livestock keepers: Livelihood transitions to 2050 in Africa due to climate change. *Environmental Science Policy*, 12 (1): 427-437.
- JONES, S., S. MURRAY, T., D. & ALLAN, R., E. 1995. The Development of Disease Resistance in Wheat. *Annual Review of Phytopathology*, 33, 429-443.
- JORDAN, V., W., L. & HUTCHEON, J., A. 2003. Influence of Cultivation Practises on Arable Crop Disease. *In: TITI, A. E. (ed.) Soil Tillage in Agro-ecosystems. USA: CRC.*
- JORGENSEN, L., N. 2008. Resistance situation with fungicides in cereals. *Zemdirbyste-Agriculture*, 95, 373-378.
- JOSHI, L., M. SINGH, D., V. & SRIVASTAVA, K., D. 1986. Wheat and wheat diseases in India. *In: Problems and Progress of Wheat Pathology in South Asia. Pp: 11-19. Malhotra Publishing House, New Delhi.*
- KAMAL, M., H. A. KIM, H., K. SHIN, H., K. CHOI, S., J. BAIK, K., B. & TSUJIMOTO, H. 2010. Abiotic stress responsive proteins of wheat grain determined using proteomics technique. *Annals Journal of Crop Science*. 4196–208.
- KEATING, B., A. CARBERRY, P., S. HAMMER, G., L. PROBERT, M., E. ROBERTSON, M., J. HOLZWORTH, D. HUTH, N., I. HARGREAVES, J., N., G. MEINKE, H. HOCHMAN, Z. MCLEAN, G. VERBURG, K. SNOW, V. DIMES, J., P. SILBURN, M. WANG, E. BROWN, S. BRISTOW, K., L. ASSENG, S. CHAPMAN, S. MCCOWN, R., L. FREEBAIRN, D., M. & SMITH, C., J. 2003. An overview of APSIM, a model designed for farming systems simulation. *European Journal of Agronomy*, 18, 267-288.
- KING, P., K. & ROBISON, L., J. 1981. ‘An interval approach to measuring decision maker preferences’. *American Journal of Agricultural Economics*, 63:510-520.
- KING, P., K. & ROBISON, L., J. 1984. ‘Risk efficiency models’. *In: Barry, P.J. (Ed.), Risk Management in Agriculture. Iowa State University Press, Ames, Iowa, 68-81.*
- KURUKULASURIYA, P., R. MENDELSON, R. HASSAN, R. BENHIN, J. DERESSA, T. DIOP, M. EID, H. YERFI-FOSU, K. GBETIBOUO, G. JAIN, S. MAHAMADOU,

- A. MANO, R. KABUBO-MARIARA, S. EL-MARSAFAWY, S. MOLUA, E. OUDA, S. OUEDRAOGO, M. SE'NE, I. MADDISON, D. NIGGOL-SEO, S. & DINAR, A. 2006. Will Africa survive climate change? World Bank Econ. Rev.
- LANDAU, S. MITCHELL, R., A., C. BARNETT, V. COLLS, J., J. CRAIGON, J. MOORE, K., L. & PAYNE, R., W. 1998. Testing winter wheat simulation models' predictions against observed UK grain yields. *Agricultural and Forest Meteorology*, 89, 85-99.
- LANDAU, S. MITCHELL, R., A., C. BARNETT, V. COLLS, J. J. CRAIGON, J. & PAYNE, R., W. 2000. A parsimonious, multiple regression model of wheat yields response to environment. *Agriculture and Forest Meteorology*. 101: 151-166.
- LEIBOVICI, D., G. MEEK, S. ANAND, S. SANTOS, R. MORLEY, J. JACKSON, M., J. MAYES, S. RAY, R. AL-AZRI, M. HODGMAN, C. BATEN, A. KING, G. BRAILSFORD, T. VU, T. KARUNARATNE, A., S. AZMAN, R. WALKER, S. AND AZAM-ALI, S. 2017. Geospatial binding for transdisciplinary research in crop science: the GRASP-GFS initiative (Geospatial Resource for Agricultural Species, pests and Pathogens with integrated workflow modelling to support Global Food Security). Open Geospatial Data, Software and Standards.
- LEROUX, P. 1998. Progress and problems in the control of cereal eyespot fungi in France. *Pesticide Outlook*, 9, 34-38.
- LIGGITT, L. JENKINSON, P. & PARRY, D., W. 1997. The role of saprophytic microflora in the development of Fusarium ear blight of winter wheat caused by *Fusarium culmorum*. *Crop Prot.* 16 (7): 679-685.
- LIU, Y. WANG, E., L. YANG, X., G. & WANG, J. 2010. Contributions of climatic and crop varietal changes to crop production in the North China Plain, since 1980s. *Global Change Biol.* 16: 2287-2299.
- LOBELL, D., B. GOURDJI, S., M. 2012. The influence of climate change on global crop productivity. *Plant physiology* 160: 1686-1697.
- LOVELL, D. POWERS, S., J. WELHAM, S., J. PARKER, S., R. 2004. A perspective on the measurement of time in plant disease epidemiology. *Plant Pathology* 53, 705-12.
- LUCAS, J., D. DYER, P., S. & MURRAY, T., D. 2000. Pathogenicity, host-specificity and population biology of *Tapesia spp.*, causal agents of eyespot disease of cereals. *Advanced in Botanical Research* 33:225-258.

- MACER, R., C. 1961. Saprophytic Colonization of Wheat Straw by *Cercospora Herpotrichoides* Fr. and Other Fungi. *Annals of Applied Biology*, **49**, 152-160.
- MADGWICK, J., W. WEST, J., S. WHITE, R., P. SEMENOV, M., A. TOWNSEND, J. A. TURNER, J. A. & FITT, B., D., L. 2011. Impacts of climate change on wheat anthesis and fusarium ear blight in the UK. *European Journal of Plant Pathology* 130: 117–131.
- MANNING, W., J. & TIEDEMANN, A. 1995. Climate change: potential effects of increased atmospheric carbon dioxide (CO₂), ozone (O₃), and ultraviolet-B (UV-B) radiation on plant diseases. *Environ Pollut.* 1995;88(2):219-45.
- MARAITE, H. DI ZINNO, T. LONGREE, H. DAUMERIE, V. & DUVEILLER, E. 1998. Fungi associated with foliar blight of wheat in warm areas. In: Duveiller E, Dubin HH, Reeves J, McNab A, eds. *Helminthosporium Blights of Wheat: Spot Blotch and Tan Spot*. Mexico D.F., Mexico: CIMMYT, 293–300
- MATUSINSKY, P. MIKOLASOVA, R. KLEM, K. & SPITZER, T. 2009. Eyespot Infection Risks on Wheat with Respect to Climatic Conditions and Soil Management. *Journal of Plant Pathology*, 91, 93-101.
- MCCOWN, R., L. & WILLIAMS, J. 1989. AUSIM: a cropping systems model for operational research. Proc. SSA ZMACS 1989 Biennial Conference on Modelling and Simulation, ANU, 25-27 Sept.
- MCCOWN, R., L. HAMMER, G., L. HARGREAVES, J., N., G. HOLZWORTH, D., P. & FREEBAIRN, D., M. 1996. A novel software system for model development, model testing and simulation in agricultural systems research. *Agric. Systems*, 50: 255-271.
- MCKEON, G., M. DAY, K., A. HOWDEN, S., M. MOTT, J., J. ORR, D., M. SCATTINI, W., J. & WESTON, E., J. 1990. Management for pastoral production in northern Australian savannas. *J. Biogeog.*, 17: 355-72.
- MEHTA, Y., E. 1996. Interdisciplinary integration-A prerequisite to integrated disease management programs. *Indian Journal Mycological Plant Pathology*, 26:178-84.
- MESSER, E. & HEYWOOD, P. 1990. Trying technology: neither sure nor soon', *Food Policy*, 15: 336-345.

- MINISTRY OF AGRICULTURE AND FISHARIES (MAF). 1993. South Batinah Integrated Study: Soil Survey and Land Classification Project OMA/87/0111. Directorate General of Agricultural Research. Sultanate of Oman.
- MISHRA, A., J., W. HANSEN, M. DINGKUHN, C. BARON, S., B. TRAORE, O. NDIAYE, & WARD, M., N. 2008. Sorghum yield prediction from seasonal rainfall forecasts in Burkina Faso. *Agriculture Forest and Meteorology*, 148:1798–1814.
- MOLDEN, D. OWEIS, Y. STEDUTO, P. KIJNE, W. HANJRA, A. BINDRABAN, S. BOUMAN, M. COOK, S. ERENSTEIN, O. FARAHANI, H. HACHUM, A. HOOGEVEEN, J. MAHOO, H. NANGIA, V. PEDEN, D. SIKKA, A. SILVA, P. TURRAL, H. UPADHYAYA, A. & ZWART, S. 2007. Pathways for increasing agricultural water productivity. In: Molden, D. (Ed.), *Comprehensive Assessment of Water Management in Agriculture, Water for Food, and Water for Life: A Comprehensive Assessment of Water Management in Agriculture*. International Water Management Institute, London: Earth scan, Colombo.
- MOORE, A., D. DONNELLY, J., R. & FREER, M. 1991. GRAZPLAN: an Australian DSS for enterprises based on grazed pastures. *Proceedings International Conference on Decision Support Systems for Resource Management*, Texas A&M University, College Station, Texas, USA, April 15-18.
- MOORE, A., D. DONNELLY, J., R. & FREER, M. 1991. GRAZPLAN: an Australian DSS for enterprises based on grazed pastures. *Proceedings International Conference on Decision Support Systems for Resource Management*, Texas A&M University, College Station, Texas, USA, April 15-18.
- MOORE, A., D. DONNELLY, J., R. & FREER, M. 1997. GRAZPLAN: decision support systems for Australian grazing enterprises. III. Pasture growth and soil moisture sub models, and the GrassGro DSS. *Agric. Syst.* 55, 535-582.
- MOORE, A., D. HOLZWORTH, D., P. HERRMANN, N., I. BROWN, H., E. DE VOIL, P., G. SNOW, V., O. ZURCHER, E., J. & HUTH, N., I. 2014. Modelling the manager: representing rule-based management in farming systems simulation models. *Environmental Modelling Software* 62, 399-410.

- MOSCHINI, G. & HENNESSY, D., A. 2001. Uncertainty, Risk Aversion, and Risk Management for Agricultural Producers. *Handbook of Agricultural Economics*, **1**, 87-153.
- MOYA-ELIZONDO, E., A. REW, L., J. JACOBSEN, B., J. HOGG, A., C. & DYER, A., T. 2011. Distribution and prévalence of Fusarium crown rot and Common root rot pathogens of wheat in Montana. *Plant Dis.* 95 :1099-1108.
- MURRAY, G. M., HEENAN, D. P. & TAYLOR, A. C. 1991. The Effect of Rainfall and Crop Management on Take-All and Eyespot of Wheat in the Field. *Australian Journal of Experimental Agriculture*, **31**, 645-651.
- MURRAY, T., D. PARRY, D., W. & CATTLIN, N., D. 2009. *Diseases of Small Grain Cereal Crops*, London: Manson Publishing.
- NARKIEWICZ-JODKO, M. GIL, Z. & LISZEWSKI, M. 2003. Effects of cultivation systems and harvest time on the health of spring barley grain. *Phytopathol. Pol.* **30**: 61–71.
- NICHOLSON, P. TURNER, A., S. & BCPC 2000. Cereal stem-base disease - a complex issue. *BCPC Conference: Pests & Diseases 2000 Proceedings*. Farnham: British Crop Protection Council. 1-3.
- NIX, J. 1979. Farm Management - State of the Art (or Science). *Journal of Agricultural Economics*, **30**, 277-292.
- NIX, J. 2010. *Farm Management Pocketbook*, Melton Mowbray: Agro Business Consultants Ltd.
- OERKE, C. & DEHNE, W. 2004. Safeguarding production - losses in major crops and the role of crop protection. *Crop Protection*. **23** (1), pp. 275-285.
- OERKE, E., C. 2006. Crop losses to pests. *Journal of Agricultural Science*, **144** (1), pp.31-43.
- O'SULLIVAN, E. DUNNE, B. KILDEA, S. & MULLINS, E. 2007. Fungicide Resistance - an increasing problem. National Tillage Conference 2007 [Online]. Available at: <http://www.teagasc.ie/publications/2007/20070131/tillageconference2007proceedings>.
- PANDEY, S. 1990. Risk efficient irrigation strategies for wheat. *Agricultural Economics*, **4**:59-71.

- PARRY, M., L. ROSENZWEIG, C. IGLESIAS, A. LIVERMORE, M. & FISCHER, G. 2004. Effects of climate change on global food production under SRES emissions and socio-economic scenarios. *Global Environmental Change* 14:53–67.
- PASSIOURA, J., B. 1996. Simulation models: Science, snake oil, education or engineering? *Agron. J.*, 88: 690–694.
- POLLEY, R., W. & CLARKSON, J., D., S. 1978. Forecasting cereal disease epidemics. In: *Plant disease epidemiology* (Eds PR Scott and A Bainbridge), pp 141-150. Blackwell Scientific Publications, Oxford.
- PREW, R., D. ASHBY, J., E. BACON, E., T., G. CHRISTIAN, D., G. GUTTERIDGE, R., J. JENKYN, J., F. POWELL, W. & TODD, A., D. 1995. Effects of incorporating or burning straw, and of different cultivation systems, on winter-wheat grown on 2 soil types, 1985-91. *Journal of Agricultural Science*, **124**, 173-194.
- RAES, D., P. STEDUTO, T., C. HSIAO, & FERERES, E. 2009. AquaCrop-The FAO crop model for predicting yield response to water: II. Main algorithms and software description. *Ag. Journal* (in press) Rosenberg, N. I. & Scott, M. J., 1994. 'Implications of policies to prevent climate change for future food security', *Global Environmental Change*.
- RAPILLY, F. 1979. Yellow Rust Epidemiology. *Annual Review of Phytopathology*, 17, 59-73.
- RAY, R., V. CROOK, M., J. JENKINSON, P. & EDWARDS, S., G. 2006. Effect of eyespot caused by *Oculimacula yallundae* and *O-acuformis*, assessed visually and by competitive PCR, on stem strength associated with lodging resistance and yield of winter wheat. *Journal of Experimental Botany*, 57, 2249-2257.
- RAY, R., V. JENKINSON, P. & EDWARDS, S., G. 2004. Effects of fungicides on eyespot, caused predominantly by *Oculimacula acuformis*, and yield of early-drilled winter wheat. *Crop Protection*, **23**, 1199-1207.
- RAEMAEKERS, R., H. 1988. Helminthosporium sativum: Disease complex on wheat and sources of resistance in Zambia. In: *Wheat Production Constraints in tropical Environments. A proceeding of the international conference, 1987.* (Ed.): A.R. Klatt. 175-186. Mexico, D.F.: CIMMYT.
- REIS, E., M. 1991. Integrated disease management-the changing concepts of controlling head blights and spot blotch. In: *Wheat in heat-stressed environments: Irrigated, dry areas*

- and rice-wheat farming systems. (Eds.): D.A. Saunders and G.P. Hettel. 165-177. Mexico, D.F.: CIMMYT.
- RICHARDSON, J. 2003, Simulation for Applied Risk Management. Program documentation. Department of Agricultural Economics, Texas A&M University.
- RICHARDSON, J., W. SCHUMANN, K. & FELDMAN, P. 2004. SIMETAR©: Simulation for Excel to analyse risk. College Station, TX: Agricultural and Food Policy Centre, Texas A&M University.
- RITCHIE, J., T. GODWIN, D., C. & OTTER, S. 1985. CERES-Wheat: A User-oriented Wheat Yield Model. Preliminary Documentation: AGRISTARS Publication No. YM-U3-04442-JSC-18892. Michigan State University, MI.
- RITCHIE, J., T. 1991. Wheat phasic development. In: Hanks, J., Ritchie, J.T. (Eds.), Modelling Plant and Soil Systems. American Society of Agronomy, Crop Science Society of America, Soil Science Society of America, Madison, Wisconsin, USA, 31-54.
- RITTER, C. DICKE, D. WEIS, M. OEBELL, H. PIEPHO, H. BUCHSE, A. & GERHARDS, R. 2008. An on farm-approach to quantify yield variation and to derive decision rules for site-specific weed management. Precision Agriculture, 9:133-146.
- ROBBERTSE, B. CAMPBELL, G., F. & CROUS, P., W. 1995. Revision of *Pseudocercospora*-like species causing eyespot disease of wheat. South African Journal of Botany 61: 43-48
- ROBERSTON, M., J. & CARBERRY, P., S., C. 2010. The evolving role of crop modelling in agronomy research. In: Food Security from Sustainable Agriculture. Edited by H. Dove and R. A. Culvenor Proceedings of 15th Agronomy Conference 2010, 15-18 November 2010, Lincoln, New Zealand.
- ROBERTSON, M., J. CARBERRY, P., S. HUTH, N., I. TURPIN, J., E. PROBERT, M., E. POULTON, P., L. BELL, M. WRIGHT, G., C. YEATES, S., J. & BRINSMEAD, R., B. 2002. Simulation of growth and development of diverse plant species in APSIM. Australian Journal of Agricultural Research 53:643-651.
- ROBINSON, J., B. FREEBAIRN, D., M. THOMAS, G., A. LAWRENCE, D., N. CAWLEY S., T. ORANGE, D., N. KING, A., J. HOLMES, C. LEHANE, K., J. DALAL, R., C. & WESTON, E., J. 2001. Can the APSIM model simulate wheat yield and grain

- protein in South-Western Queensland?. Crop Modelling and Management. 10th Australian Agronomy Conference.
- ROTTER, R., P. CARTER, T., R. OLESEN, J., E. & PORTER, J., R. 2011. Crop-climate models need an overhaul. *Nature Climate Change* 1:175-177.
- ROWE, R., C. & POWELSON, R., L. 1973. Epidemiology of *Cercospora* foot rot of wheat: Disease spread. *Phytopathology* 63: 984-988.
- SANCHEZ, P., A. SHEPHERD, K., D. SOULE, M., J. PLACE, F., M. BURESH, R., J. IZAC, A., M. MOKWUNYE, A., U. KWESIGA, F., R. NDIRITU, C., G. & WOOMER, P., L. 1997. Soil fertility replenishment in Africa: and investment in natural resource capital. *Replenishing soil fertility in Africa*. 51:1-46.
- SANSFORD, C., E. BAKER, R., H., A. BRENNAN, J., P. EWERT, F. GIOLI, B. INMAN, A. KINSELLA, A. MAGNUS, H., A. MIGLIETTA, F. MURRAY, G., M. PORTA-PUGLIA, A. PORTER, J., R. RAFOSS, T. RICCIONI, L. & THORNE, F. 2008. The new Pest Risk Analysis for *Tilletia indica*, the cause of Karnal bunt of wheat, continues to support the quarantine status of the pathogen in Europe. *Plant Pathology* 57: 603-611.
- SCHILDER, A. & BERGSTROM, G., C. 1993. Tan spot (*Pyrenophora tritici-repentis*). In: *Seed-borne diseases and seed health testing of wheat*. (Eds.): S.B. Mathur and B.M. Cunfer. 113-122. Danish.
- SCHOLES, R., J. & BIGGS, R. 2004. *Ecosystem Services in Southern Africa: A Regional Assessment*. Pretoria, South Africa: Council for Scientific and Industrial Research.
- SCHULTZ, B. & DE WRACHIEN, D. 2002. Irrigation and drainage systems research and development in the 21st century. *Irrigation and drainage* 51 (4): 311-327.
- SCOTT, P., R. 1971. The effect of temperature on eyespot *Cercospora herpotrichoides* in wheat seedling. *Annals of Applied Biology* 68:169-175.
- SCOTT, P., R. & HOLLINS, T., W. 1974. Effects of Eyespot on Yield of Winter-Wheat. *Annals of Applied Biology*, **78**, 269-276.
- SCOTT, P., R. HOLLINS, T., W. & MUIR, P. 1975. Pathogenicity of *Cercospora herpotrichoides* to wheat, barley, oats and rye. *Transactions of the British Mycological Society*, 65, 529-538.

- SCOTT, P., R. & HOLLINS, T. W. 1978. Prediction of Yield Loss Due to Eyespot in Winter-Wheat. *Plant Pathology*, 27, 125-131.
- SEMENOV, M., A. & SHEWRY P., R. 2011. Modelling Predicts that Heat Stress, not Drought, will Increase Vulnerability of Wheat in Europe Scientific Reports. 1:66.
- SHAZIA, I. & IFTIKHAR, A. 2005. Prevalence and disease incidence of foliar blight of wheat in rice wheat cropping system of Punjab. *Pak. J. Bot.*, 37(4):973-980.
- SHENG, H. SEE, D. & MURRAY, T., D. 2012. Mapping QTL for resistance to eyespot of wheat in *Aegilops longissima*. *Theoretical and Applied Genetics*, 5, 1-12.
- SELING, K. STAHL, C. WINKELMANN, C. & CHRISTEN, O. 2005. Growth and yield of winter wheat in the first 3 years of a monoculture under varying N fertilization in NW Germany. *European Journal of Agronomy*, 22, 71-84.
- SILVIA, P. & RUTH, Dill-Mackey. 2010. Fusarium Species Recovered from Wheat and Barley Grains in Uruguay, Pathogenicity and Deoxynivalenol Content. *Agrociencia Uruguay*. 14(2):33- 44.
- SINCLAIR, T., R. & MUCHOW, R., C. 1999. Radiation use efficiency. *Advances in Agronomy*. 65:215-265.
- SINGH, D., V. & SRIVASTAVA, K., D. 1997. Foliar blights and Fusarium scab of Wheat. In: Management of Threatening Plant Diseases of National Importance: Present status and Strategies for Management (Eds. Agnihotry, V.P., Sarabhoy, A.K. and Singh, D.V.). Malhotra Publishing House, New Delhi. pp. 1-16.
- SINGH, R., N. SINGH, A., K. SHIV, P., S. & SINGH, B., N. 2001. Prevalence and distribution of foliar blight pathogens attacking wheat in India. *Indian Phytopath.* 54(2):175-178.
- SINGH, R., P. KINYUA, M., G. WANYERA, R. NJAU, P. Jin Y. & HUERTA-ESPINO, J. 2007. Spread of a highly virulent race of *Puccinia graminis tritici* in eastern Africa.
- SMIL, V. 2000. Feeding the World: A Challenge for the 21st Century. The MIT Press, Cambridge, MA, 360 pp.
- SMILEY, R., W. GOURLIE, J., A. EASLEY, S., A. & PATTERSON, L., M. 2005. Pathogenicity of fungi associated with the wheat crown rot complex in Oregon and Washington. *Plant Dis* 89:949–957.

- SMILEY, R., W. 2009. Water and Temperature Parameters Associated with Winter Wheat Diseases Caused by Soil borne Pathogens. *Plant Disease*, **93**, 73-80.
- SMITH, F., P. HOLZWORTH, D., P. & ROBERTSON, M., J. 2005. Linking icon-based models to code-based models: a case study with the agricultural production systems simulator. *Agric. Syst.* 83: 135-151.
- SMITH, J., M. COOK, S., K. MILLS, A., R. BACON, E., T., G. & CLARKE, J., H. 2000. The effect of three or five years of set-aside on the husbandry and grain yield of subsequent cereal crops in the UK. *Plant and Soil*, **225**, 279-297.
- SOULIE, M., C. VIAN, B. & GUILLOTSALOMON T. 1985. Host-parasite interactions during infection by *Cercospora-herpotrichoides*, an agent of root-rot - morphology parasite and ultrastructure of the walls of sensitive and resistant hosts. *Canadian Journal of Botany* 63:851-858
- STAPPER, M. FISCHER, R., A. 1990. Genotype, sowing date and plant spacing influence on high yielding irrigated wheat in southern New South Wales. II. Growth, yield and nitrogen use. *Australian Journal of Agricultural Research*: 41, 1021-1041.
- STEDUTO, P. HSIAO, T., C. RAES, D. & FERERES, E. 2009. AquaCrop- The FAO crop model to simulate yield response to water, 1. Concepts and underlying principles. *Agronomy Journal* 101:426-437.
- STRANGE, R., N. & SCOTT, P., R. 2005. Plant disease: a threat to global food security. *Annu Rev Phytopathology* 43:83-116.
- STRAUSBAUGH, C., A. BRADLEY, C., A. KOEHN, A., C. & FORSTER, R., L. 2004. Survey of root diseases of wheat and barley in south eastern Idaho. *Canadian Journal of Plant pathology*. 26(2):167-176.
- SUFFERT, F. & SACHE, I. 2011. Relative importance of different types of inoculum to the establishment of *Mycosphaerella graminicola* in wheat crops in north-west Europe. *Plant Pathology*, **60**, 878-889.
- SUTHERST, R. BAKER, R., H., A. COAKLEY, S., M. HARRINGTON, R. KRITICOS, D., J. & SCHERM, H. 2007. Pests under global change: meeting your future landlords? In: Canadell, J. G. editor. *Terrestrial ecosystems in a changing world*. Berlin: Springer; 211-225.

- SUTHERST, R., W. & MAYWALD, G., F. 1998. DYMEX modelling workshops: a national, collaborative approach to pest risk analysis and IPM in Australia. *Pest Management Future Challenge*. 2:57-62.
- TAMURA, K. STECHER, G. PETERSON, D. FILIPSKI, A. & KUMAR, S. 2013. MEGA6: Molecular Evolutionary Genetics Analysis Version 6.0. *Molecular. Biology. Evolution*. 30 (12): 2725–2729.
- THORNBY, D., F. & WALKER, S., R. 2009. Simulating the evolution of glyphosate resistance in grains farming in northern Australia. *Ann. Bot.* 104:747-756.
- THORNBY, D., F. WALKER, S., R. & WHISH, J., P., M. 2010. Modelling to Estimate Glyphosate Resistance Risk in Barnyard Grass in the Northern Australian Grain Region. *Australian Weed Science Society*. 1-3.
- THRANE, U. 2001. Developments in the taxonomy of *Fusarium* species based on secondary metabolites. Pages 29-49 in: *Fusarium*. Paul E. Nelson memorial symposium. B. E. Summerell, Leslie, J. F. D., Backhouse, W. L., and Bryden, L. W. Burgess, eds. The American Phytopathological Society Press. St. Paul, MN.
- TONEV, T., K. KIRYAKOVA, V. & MILEV, G. 2008. Influence of some agronomy factors on spike components after a rare incidence of fusarium head blight epiphytoty of winter wheat, I. Effect of longterm crop rotation, mineral fertilization and sowing term, *Bulg. J. Agric. Sci.*,14: 321-328
- TUNALI, B. NICOL, J., M. HODSON, D. UCKUN, Z. BUYUK, O. ERDURMUS, D. HEKIMHAN, H. AKTAS, H. AKBUDAK, M. & BAGCI, A. 2008. Root and crown rot fungi associated with spring, facultative and winter wheat in Turkey. *Plant Dis* 92:1299–1306.
- TURKINGTON, T., K. KUZYK, A. & DUNN, R. 2004. "Irrigation and plant disease management,"
[http://www1.agric.gov.ab.ca/\\$department/deptdocs.nsf/all/ind10759/\\$file/irrigation](http://www1.agric.gov.ab.ca/$department/deptdocs.nsf/all/ind10759/$file/irrigation).
- TURNER, A., S. NICHOLSON, P. EDWARDS, S., G. BATEMAN, G., L. MORGAN, L., W. TODD, A., D. PARRY, D., W. MARSHALL, J. & NUTTALL, M. 2001. Evaluation of diagnostic and quantitative PCR for the identification and severity assessment of eyespot and sharp eyespot in winter wheat. *Plant Pathology*, **50**, 463-469.

- UNITED KINGDOM CLIMATE IMPACTS PROGRAMME. 2011. Thinking Climate—Putting Scientific Knowledge into the Heart of Decision-making. Available: <http://www.ukcip.org.uk/index.php>.
- VAN DER MENSBRUGGHE, D. OSORIO-RODARTE, I. BURNS, A. & BAFFES, J. 2009. Macroeconomic environments, commodity markets: A longer term outlook. Paper prepared for the Expert Meeting on How to Feed the World in 2050, FAO Headquarters, Rome. Food and Agriculture Organization of the United Nations. Economic and Social Development Department. The World Bank, 1818 H Street, NW, Washington, D.C. 20433, United States of America.
- VON BRAUN, J. 2007. The world food situation: new driving forces and required actions. Washington, DC, USA: International Food Policy Research Institute.
- WALKER, B. & STEFFEN, W. 1997. An overview of the implications of global change for natural and managed terrestrial ecosystems. Conservation Ecology [online]1(2): 2. Available from the Internet. URL: <http://www.consecol.org/vol1/iss2/art2/>.
- WALKER, P., T. 1983. Crop losses: the need to quantify the effects of pests, diseases and weeds on agricultural production. Agric. Ecosystems Environment. 9: 119-158.
- WAN, A., M. BOCK, C., H. FITT, B., D., L., HARVEY, J., L. & JENKYN, J., F. 2005. Development of *Oculimacula yallundae* and *O. acuformis* (eyespot) on leaf sheaths of winter wheat in the UK in relation to thermal time. Plant Pathology 54, 144-155.
- WANG, J. WANG, E. & LIU, D., L. 2011. Modelling the impacts of climate change on wheat yield and field water balance over the Murray-Darling Basin in Australia. Theoretical and Applied Climatology 104:285-300.
- WEISZ, R. COWGER, C. AMBROSE, G. & GARDNER, A. 2011. Multiple Mid-Atlantic Field Experiments Show No Economic Benefit to Fungicide Application When Fungal Disease Is Absent in Winter Wheat. *Phytopathology*, 101, 323-333.
- WELHAM, S., J. TURNER, J., A. GLADDERS, P. FITT, B., D., L. EVANS, N. & BAIERL, A. 2004. Predicting light leaf spot (*Pyrenopeziza brassicae*) risk on winter oilseed rape (*Brassica napus*) in England and Wales, using surveys, weather and crop information. Plant Pathology 53, 713-724.
- WEST, J. TOWNSEND, J. STEVENS, M. & FITT, B. 2012. Comparative biology of different plant pathogens to estimate effects of climate change on crop diseases in Europe. European Journal of Plant Pathology. 133: 315-331.

- WEST, S., J., E. BOOTH, G., M. BECK, J., J. & ETIENNE L. 1998. A survey of *Tapesia yellundae* and *Tapesia acuformis* in UK winter wheat crops using a polymerase chain reaction diagnostic assay. In: *Proceedings of the Brighton Crop Protection Conference*. British Crop Protection Council, Farnham, UK, pp. 1029-1034.
- WHEELER, T. & VON BRAUN, J. 2013. Climate change impacts on global food security. *Science* Vol. 341 no. 6145: 508-513.
- WHISH, J., P., M. HERMANN, N., I. WHITE, N., A. MOORE, A., D. & KRITICOS, D., J. 2015. Integrating pest population models with biophysical crop models to better represent the farming system. *Environmental Modelling and Software*. 72:418-425.
- WHITE, N., A. CHAKRABORTY, S. & MURRAY, G., M. 2004. A Linked Process-based Model to Study the Interaction between *Puccinia striiformis* and Wheat. In: Presented at the 11th International Crop Science Congress, Brisbane, Australia.
- WIIK, L. 2009. Yield and disease control in winter wheat in southern Sweden during 1977–2005. *Crop Protection*, **28**, 82-89.
- WOOD, J. 2011. Malthus, Famine, Disease and World Population. <http://www.thenevadaview.com/2264/malthus-famine-disease-andworld-population/html>
- WOSSEN, T. BERGER, T. & DI FALCOC, S. 2015. Social capital, risk preference and adoption of improved farm land management practices in Ethiopia. *Agricultural Economics* 46:81–97.
- YARHAM, D., J. 1986. Change and decay - the sociology of cereal foot rots. British. Crop Protection Conference Pests and Diseases Vol 2. British Crop Protection. Council, pp 401-410.
- ZADOKS, J., C. CHANG, T., T. & KONZAK, C., F. 1974. A décimale code for the growth stage of cereals. *Weed Research* 14:415-421.
- ZHANG, Y., I. FENG, L. WANG, E. WANG, J. & LI, B. 2012. Evaluation of the APSIM-Wheat model in terms of different cultivars, management regimes and environmental conditions. *Canadian Journal of Plant Science* 92: 937-949.

7. APPENDIX

Table 7-1 Historical data used in this study collected through previous research projects on fungicide efficacy against eyespot disease by the University of Nottingham, Harper Adams University, as well as The Arable Group research (TAG).

Year	Trial code	County	Region	Tillage	Actual sow date	Variety name	Soil type	Rotation
2004	LO15r	Shropshire	West	Ploughed	03/10/2003	Einstein	Sandy Loam	1st WW
2004	LO15w	Shropshire	West	Ploughed	03/10/2003	Einstein	Sandy Loam	1st WW
2004	LO14r	Shropshire	West	Ploughed	03/10/2003	Einstein	Sandy Loam	1st WW
2004	LO14w	Shropshire	West	Ploughed	03/10/2003	Einstein	Sandy Loam	1st WW
2005	MO17r	Shropshire	West	Ploughed	05/10/2004	Gladiator	Sandy Loam	1st WW
2005	MO17w	Shropshire	West	Ploughed	05/10/2004	Gladiator	Sandy Loam	1st WW
2005	MO18r	Shropshire	West	Ploughed	05/10/2004	Gladiator	Sandy Loam	1st WW
2005	MO18w	Shropshire	West	Ploughed	05/10/2004	Gladiator	Sandy Loam	1st WW
2006	NO16r	Shropshire	West	Ploughed	19/09/2005	Robigus	Sandy Loam	1st WW
2006	NO16w	Shropshire	West	Ploughed	19/09/2005	Robigus	Sandy Loam	1st WW
2006	NO25r	Shropshire	West	Ploughed	19/09/2005	Robigus	Sandy Loam	1st WW
2006	NO25w	Shropshire	West	Ploughed	19/09/2005	Robigus	Sandy Loam	1st WW

APPENDIX I

2007	PO12	Shropshire	West	Ploughed	02/10/2006	Robigus	Sandy Loam	1st WW
2007	PO13	Shropshire	West	Ploughed	19/09/2006	Alchemy	Sandy Loam	1st WW
2007	PO14	Shropshire	West	Ploughed	02/10/2006	Robigus	Sandy Loam	1st WW
2007	PO15	Shropshire	West	Ploughed	19/09/2006	Alchemy	Sandy Loam	1st WW
2007	PO16	Shropshire	West	Ploughed	19/09/2006	Alchemy	Sandy Loam	1st WW
2007	PO17	Shropshire	West	Ploughed	02/10/2006	Robigus	Sandy Loam	1st WW
2008	RO21	Shropshire	West	Ploughed	11/10/2007	Gladiator	Clay Loam	1st WW
2008	RO22	Shropshire	West	Ploughed	11/10/2007	Gladiator	Clay Loam	1st WW
2008	RO26	Shropshire	West	Ploughed	18/10/2007	Timber	Sandy Loam	1st WW
2009	N09r	Leicestershire	East	Min-till	01/10/2008	Robigus	Clay Loam	1st WW
2009	N09z	Leicestershire	East	Min-till	01/10/2008	Zebedee	Clay Loam	1st WW
2009	BASFW	Leicestershire	East	Min-till	03/10/2014	Einstein	Clay Loam	Grass
2009	BASFR	Leicestershire	East	Min-till	03/10/2014	Einstein	Clay Loam	Grass
2010	O10p	Leicestershire	East	Ploughed	27/10/2009	Panorama	Clay Loam	1st WW
2010	O10z	Leicestershire	East	Ploughed	27/10/2009	Zebedee	Clay Loam	1st WW
2010	BASF Product	Leicestershire	East	Ploughed	02/10/2009	Cordiale	Clay Loam	1st WW

APPENDIX I

2010	BASF	Leicestershire	East	Ploughed	02/10/2009	Cordiale	Clay	1st WW
	Dose						Loam	
2011	HGCA	Leicestershire	East	Ploughed	11/10/2010	Gallant	Clay	1st WW
	Timing						Loam	
2011	Syngenta	Leicestershire	East	Ploughed	11/10/2010	Gallant	Clay	1st WW
							Loam	
2011	BASF	Shropshire	West	Ploughed	30/09/2010	Panorama	Sandy	1st WW
	Product						Loam	
2011	BASF	Shropshire	West	Ploughed	30/09/2010	Panorama	Sandy	1st WW
	Dose						Loam	
2012	Eyespot in	Leicestershire	East	Ploughed	26/09/2011	Robigus	Clay	Winter
	Oakley						Loam	Oat
2012	BASF	Leicestershire	East	Ploughed	05/10/2011	2nd wheat	Clay	1st WW
	Dose						Loam	
2012	BASF	Leicestershire	East	Ploughed	05/10/2011	2nd wheat	Clay	1st WW
	Product						Loam	
2013	BASF	Leicestershire	East	Ploughed	03/10/2012	Scout	Clay	1st WW
	Trials						Loam	
2013	Eyespot in	Leicestershire	East	Ploughed	19/09/2012	Oakley	Clay	1st WW
	Oakley						Loam	
2014	BASF	Shropshire	East	Ploughed	01/10/2013	JB Diego	Clay	1st WW
	Product						Loam	

Table 7-2 Experimental field locations and their GPS coordination's

County	Field Location	Latitude	Longitude
Shropshire	Bayely Hills,HAUC, Newport	52.77063	-2.409847
	Sidlington Field, Newport	52.789008	-2.437077
	30 acre, Newport	52.79486	-2.446175
	Sambrook, Newport	52.816354	-2.428858
	Furniss, Newport	52.797896	-2.433536
	Garden Field, Newport	52.778639	-2.430317
	Upperwood Leasow field, Harper	52.778885	-2.426741
	Adams Farm,		
	B1 Field, Sutton Bonington, East	52.836726	-1.24681
	Midlands		
Leicestershire	B2 Field, Sutton Bonington, East	52.839811	-1.245141
	Midlands		
	Bunny Field 2, Sutton Bonington,	52.856071	-1.128098
	East Midlands		
	Watton Estate Field, Sutton	52.836726	-1.24681
	Bonington, East Midlands		
	S24 Field, Sutton Bonington, East	52.836726	-1.24681
	Midlands		
	S31 Field, Sutton Bonington, East	52.839811	-1.245141
	Midlands		

Table 7-3 Trials of eyespot disease inoculated and natural infection between 2004 and 2014.

Year	Location	Inoculation/Natural infection
2004	Bayely Hills,HAUC, Newport, Shronshire	W+R eyespot species inoculation
2005	Sidlington Field, Newport, Shropshire	W+R eyespot species inoculation
2006	30 acre, Newport, Shropshire	W+R eyespot species inoculation
2007	Sambrook, Newport, Shropshire	Natural infection
2007	Furniss, Newport, Shropshire	W+R Species inoculation
2008	Garden Field, Newport, Shropshire	W+R eyespot species inoculation
2008	Furniss, Newport, Shropshire	Natural infection
2009	Bunny field 2, Sutton Bonington, East Midlands	Natural infection
2009	Bunny field 2, Sutton Bonington, East Midlands	W+R eyespot species inoculation
2010	B1 field, Sutton Bonington, East Midlands	Natural infection
2010	B1 field, Sutton Bonington, East Midlands	W+R eyespot species inoculation
2011	S31 field, Sutton Bonington, East Midlands	W+R eyespot species inoculation
2012	S24 & Watton Estate fields, Sutton Bonington, East Midlands	W+R eyespot species inoculation
2013	B1 and B2 fields, Sutton Bonington, East Midlands	W+R eyespot species inoculation
2014	Upperwood Leasow field, Harper Adams Farm, Shropshire	W+R eyespot species inoculation

Table 7-4 Different fungicides products and their active ingredients used in the trials.

Active Ingredients	Trade Name	Manufacturer
epoxiconazole	Opus	BASF plc
Metrafenone	Flexity	BASF plc
prothioconazole	Proline 275	Bayer Crop Science Limited
boscalid and epoxiconazole	Tracker	BASF plc
cyprodinil	Unix	Syngenta Crop Protection UK Limited
epoxiconazole and prochloraz	Ennobe	BASF plc
epoxiconazole and fluxapyroxad	Adexar	BASF plc
azoxystrobin	Amistar	Syngenta Crop Protection UK Limited
bixafen and prothioconazole	Aviator 235Xpro	Bayer Crop Science Limited Syngenta UK Limited
chlorothalonil	Bravo	BASF plc
epoxiconazole, fenpropimorph and metrafenone	Capalo	BASF plc
epoxiconazole, fluxapyroxad and pyraclostrobin	Cerix	BASF plc
boscalid and epoxiconazole	Chord	BASF plc
boscalid and epoxiconazole	Enterprise	BASF plc
epoxiconazole	Ignite	BASF plc
fluxapyroxad	Imtrex	BASF plc
fluxapyroxad and metconazole	Librax	BASF plc
boscalid, epoxiconazole and pyraclostrobin	Nebula	BASF plc
epoxiconazole and Isopyrazam	Seguris	Syngenta UK Limited
Penthiopyrad	Vertisan	Du Pont (UK) Limited
fluxapyroxad	Xemium	BASF plc

8. APPENDIX

8.1. Agriculture and Climate Change - Adapting Crops to Increased

Uncertainty (AGRI 2015 Conference)

Simulating eyespot disease development and yield loss using APSIM for UK wheat

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A Global crop production is affected by seasonal and climatic variations in temperature, rainfall patterns or intensity and the occurrence of abiotic and biotic stresses. Climate change can alter pest and pathogen populations as well as pathogen complexes that pose an enormous risk to crop yields and future food security. Eyespot disease caused by *Oculimacula yallundae* and *O. acuformis* is associated with yield losses in UK wheat estimated in 1998 at £24 million. Crop simulation models have been validated as an important tool for the development of more resilient agricultural systems and improved decision making for growers. The Agricultural Production Systems Simulator (APSIM) is a software tool that enables sub-models to be incorporated for simulation of production in diverse agricultural systems. APSIM-wheat simulates crop growth and development, soil and management options. Modification of APSIM to incorporate epidemiological disease model for crop growth and yield under different disease intensities has not yet been undertaken in UK or elsewhere. Thus, the objective of this work was to develop epidemiological model for eyespot disease and incorporate it within APSIM for crop simulation under a range of disease and environmental conditions. Historical climatic data combined with 8 years of observed disease (2004-2012) data on incidence and severity of eyespot in UK field trials was used to develop epidemiological model, combining infection and severity, for the prediction of disease development in relation to crop growth stages. Crop growth characteristics, biomass and yield were measured separately and employed for eyespot yield loss or biomass reduction model in wheat based on disease severity. Current work is focused on modifying APSIM to simulate crop loss through the incorporation of the epidemiological disease and yield reduction components and further validation to confirm that empirical data were accurately simulated.

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Keywords: *Oculimacula yallundae*, *O. acuformis*, APS IM

References

- 1- Bock, A. M., Wan, A. M., & Fitt, B. D. L. 2009. Development of *Oculimacula yallundae* and *O. acuformis* (eyespot) lesions on stems of winter wheat in relation to thermal time in the

UK. Journal of Plant Pathology, 58: 12-22.

2- McCown, R. L., Hammer, G. L., Hargreaves, J. N. G., Holzworth, D. P. & Freebairn, D. M. 1996. A novel software system for model development, model testing and simulation in agricultural systems research. Agric. Systems, 50: 255-271.

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